Annals of Otology, Rhinology and Laryngology

FOUNDED BY JAMES PLEASANT PARKER

INCORPORATING
THE INDEX OF OTOLARYNGOLOGY

H. W. LOEB, M. D., Editor-in-Chief

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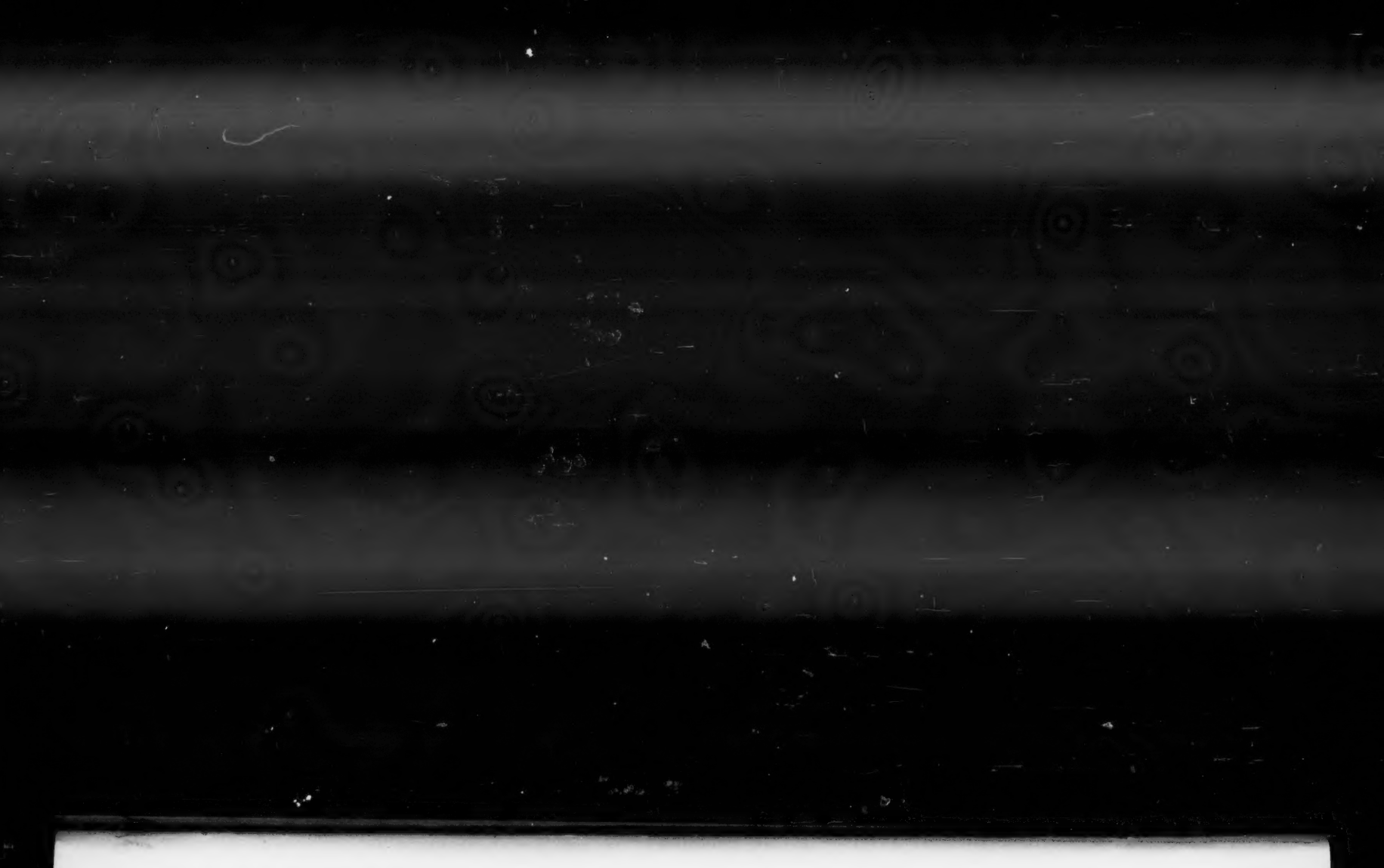

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VOL. XXXIV. MARCH, 1925. NO. 1.

I.

PROGRESS IN AMERICAN OTOLARYNGOLOGY.

BY HANAU W. LOEB, M. D.,
ST. LOUIS.

Significant evidence of the progress of otolaryngology in America has been given by the establishment of the American Board of Otolaryngology and the foundation of the Archives of Otolaryngology.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

The incorporation of the American Board of Otolaryngology in January of this year resulted from the persistent activity of Dr. George E. Shambaugh of Chicago, who for many years has labored to secure the support of the national otolaryngological associations. His efforts became successful when the first Board was appointed by the five associations, as follows: Dr. T. H. Halsted of Syracuse and Dr. H. W. Loeb of St. Louis representing the American Otological Society; Dr. H. P. Mosher of Boston and Dr. R. H. Skillern of Philadelphia, the American Laryngological Association; Dr. B. R. Shurly of Detroit and Dr. F. R. Spencer of Boulder, Colorado, the American Laryngological, Rhinological and Oto-

logical Society; Dr. T. W. Carmody of Denver and Dr. W. P. Wherry of Omaha, the American Academy of Ophthalmology and Otolaryngology; and Dr. J. C. Beck of Chicago and Dr. R. C. Lynch of New Orleans, the Section of Laryngology, Otology and Rhinology of the American Medical Association.

The first meeting of the Board was held on November 10, 1924, the following officers being elected:

Dr. Harris P. Mosher, Boston, President

Dr. Frank R. Spencer, Boulder, Vice-President.

Dr. Hanau W. Loeb, St. Louis, Secretary and Treasurer.

The chief activities announced by the Board are as follows:

First. To establish standards of fitness to practice Otolaryngology.

Second. To investigate and prepare lists of medical schools, hospitals and private instructors recognized as competent to give the required training in Otolaryngology.

Third. To arrange, control and conduct examinations to test the qualifications of those who desire to practice Otolaryngology and to confer a certificate upon those who meet the established standards.

These activities proceed from the object of the Corporation, which is stated in the Articles of Incorporation to be:

"The object of the corporation shall be to elevate the standard of Otolaryngology, to familiarize the public with its aims and ideals, to protect the public against irresponsible and unqualified practitioners, to receive applications for examination in Otolaryngology, to conduct examinations of such applicants, to issue certificates of qualification in Otolaryngology and to perform such duties as will advance the cause of Otolaryngology."

The Board has already received more than three hundred applications.

In determining the question of certification, the examiners rely on the following criteria:

First. The applicant's professional record.

Second. Written reports of the prescribed number of cases that he has observed and treated.

Third. A practical, clinical, and laboratory examination, when required.

Fourth. A written examination, when required.

The Board announces that every effort will be made to avoid a type of examination which can be passed by cramming, as it is not desirable to test exclusively nor even primarily the memory of the candidate. Hence the stress on the written examination is less than might be expected.

The subdivisions of the practical examination are:

First. Examination of the auricle, external auditory meatus and membrana tympani; functional tests of hearing with the tuning forks, Galton whistle, etc.; turning tests, caloric tests and tests for past pointing. Other and more delicate tests may be required in some cases, if at the discretion of the Board or of the individual examiner this seems desirable to test the candidate's fitness, depending upon his training and experience in Otolaryngology.

Second. Examination of the nose by anterior rhinoscopy, examination of the nasopharynx by posterior rhinoscopy and examination of the larynx by indirect laryngoscopy; transillumination of the accessory nasal sinuses; direct laryngoscopy with a laryngeal spatula or by suspension laryngoscopy; examination with an electric pharyngoscope and perhaps other examinations, if in the judgment of the Board or the individual examiner this seems desirable.

Third. The relation of diseases of the ear, nose and throat to conditions and diseases in other parts of the human body.

Fourth. Therapeutics.

Fifth. Surgery of the ear, nose and throat.

Sixth. Laboratory examination.

The policy of the Board at present is to place chief emphasis on the ability to draw correct inferences and make intelligent use of reports from pathologists, rather than an ability personally to carry out all laboratory manipulations and examinations.

Something of the latter will be required of the most recent graduates, who may be asked to examine specimens, identify tissues and microorganisms, and interpret pathologic conditions. As the teaching of Otolaryngology improves, more of this practical laboratory work will be required, including work in anatomy, physiology, bacteriology and pathology.

The written examination is intended to cover such of the underlying principles or science of Otolaryngology as cannot be covered by the practical examinations. It will include

anatomy, embryology, physiology, pathology and relations of the ear, nose and throat to other organs and diseases of the body. After a few years, more stress may be laid on fundamental principles. It is not intended to make the examinations in the beginning more severe than is warranted by the present available facilities for the study of this subject. However, it is one of the chief aims of the Board to improve the teaching of Otolaryngology. No one can deny that, unless its foundations are laid broad and deep in the underlying sciences and principles, the superstructure of clinical Otolaryngology in America must always remain incomplete.

ARCHIVES OF OTOLARYNGOLOGY.

For more than twenty years efforts have been made to secure the cooperation of the American Medical Association in the publication of a scientific journal devoted to Otolaryngology. These efforts have been brought to a successful issue by the establishment of the Archives of Otolaryngology, the first number of which appeared in January of this year. This journal, which is a welcome addition to the other important American and English laryngologic journals, gives great scientific promise to American Otolaryngology.

The original articles are to be of the highest class. Regular reviews will appear monthly and collated reviews from time to time. An endeavor will be made to have the society reports and news items as complete as possible.

Under the guidance of Dr. Shambaugh and his corps of editors there is every reason to believe that the Archives of Otolaryngology will take a place among the highest of the world's publications in our field and will reflect great credit upon American Otolaryngology.

11.

FOREIGN BODIES IN THE AIR AND FOOD PASSAGES.*

By Gabriel Tucker, M. D., and Louis H. Clerf, M. D.,

Philadelphia.

All clinicians agree as to the value of experience as a guide to the practice of medicine and surgery. To be of value, however, it is necessary that the experience be available at command when needed. Unfortunately, there is a limit to the average human memory. Therefore, it cannot always measure fully up to requirements in recalling for use all the past experience, however vividly certain outstanding cases may loom. For quickly reviewing past experience in probably similar cases the value of tabulated records, specially arranged with such an object in view, has been stated by Chevalier Jackson.†

The object of the present paper is to present in similar form the cases that have come to the Bronchoscopic Clinic since Dr. Jackson's previous publications.‡

In about 20 per cent of the cases, removal had been attempted by other bronchoscopists, in some instances as many as four times. All mention of this has been omitted. This previous instrumentation accounted for the pathology present and also for the complicated mechanical problem in many instances, and it increased our mortality.

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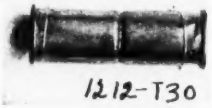
*From the Bronchoscopic Clinic, Philadelphia.

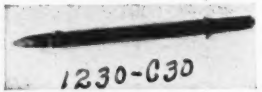
†Charted Experiences in Cases at the Bronchoscopic Clinic, Research Fund Prize Essay, Proceedings Am. Laryngol., Rhinol. and Otol. Soc., 1923; also Annals of Otol., Rhinol. and Laryngol., 33:1009, 1924.

‡Ibid.; also, Observations on the Pathology of Foreign Bodies in the Air and Food Passages, Mütter Lecture, Surg. Gynecol. and Obst., March, 1919; also Peroral Endoscopy and Laryngeal Surgery Textbook, 1914.

HARDWARE

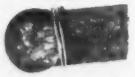
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1156	11 yrs.	Wire shaped like fish hook	Left main bronchus, 2 years	None	7 mm., aspirating	Fish hook shaped object	Side-curved	Curved end after version	Extraction Cure	3 min. 30 sec.	Oral	Large granuloma projecting into lumen of left bronchus from anterior wall. Bloody pus coming up from below; disappeared out through bronchoscopic aspirating tube. Long branch of foreign body visible in swollen mucosa on external wall of left bronchus. Foreign body grasped by short end and slid upward to bifurcation where it was turned long end down into right bronchus, the curve brought upward after re-grasping.
Fbdy. 1191	3 yrs.	Tack	Right lower lobe bronchus, 2 months	None	5 mm. x 30 cm., standard	Buried point	Tucker	Shaft after disengaging	Extraction Cure	5 min. 45 sec.	Oral	Shaft of tack buried up to head. Head grasped and pushed down until point emerged; then shaft grasped.
Fbdy. 1212 T-30 	11 yrs.	Pencil cap	Right main bronchus, 2 days	None	6 mm. x 35 cm., standard	Covering free edge of cylindrical object with lip of tube to prevent stripping off at the glottis	Forward grasping	Inner edge	Extraction Cure	2 min. 55 sec.	Oral	Larynx and trachea slightly reddened. The uppermost end of foreign body was found in right main bronchus just below level of carina. Lumen of foreign body closed with thick pus.

(1)

Fbdy. 1230 C-30  	14 yrs.	Nail	Left lower lobe bronchus, internal branch, 7 days	None	6 mm. x 35 cm., standard	Finding nail in bronchus too small for bronchoscope to enter	Forward grasping	Point	Extraction Cure	8 min. 55 sec.	Oral	No pathology visible. Double-plane fluoroscopic guidance of Dr. W. F. Manges required for finding of invaded bronchus.
Fbdy. 1235 T-33 	2 yrs.	Metal key	Esophagus just below cricopharyngeus, 4 hours	None	5 mm. x 30 cm., standard	Rotation of object to pass the cricopharyngeus	Rotation	Ring in left end of handle of foreign body	Extraction Cure	6 min. 15 sec.	Oral	Handle end of foreign body upward, long axis of its upper end was in lateral plane of esophagus. On inspiratory efforts it would disappear behind cricopharyngeus.
Fbdy. 1237 	5 yrs.	Carpet tack	Right inferior lobe bronchus, 1½ years	None	6 mm. x 35 cm., standard	Tack head up below stricture	Plain with cupped faces after dilating stricture	Head of tack	Extraction Cure	21 min. 10 sec. 5 min. 7 sec.	Oral	First bronchoscopy: Right bronchus found strictured just below orifice of middle lobe. Portion of head of tack visible below stricture. Tack grasped with plain cupped forceps but lumen of stricture not sufficiently large to permit removal without trauma. Stricture dilated. Pus aspirated. Second bronchoscopy (one week later): Tack found in inferior lobe bronchus below a tight stricture. Lumen of stricture considerably larger than at last examination, having evidently retained a large portion of dilatation then produced with dilating forceps. Head of tack seized with plain forceps having cupped faces and removed. Fluoroscopic assistance by Dr. Manges.

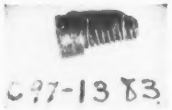
HARDWARE—*(Continued)*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1243	6 yrs.	Screw	Right bronchus below orifice of middle lobe, 2½ yrs. (approximately)	None	6 mm. x 35 cm., standard	Strictured bronchus above bronchiectatic abscess	Plain with cupped faces	Head	Extraction Cure	27 min. 45 sec.	Oral	Stricture found in right bronchus just below orifice of middle lobe bronchus. Stricture dilated by working through it with forceps until the stricture would permit the passage of the bronchoscope through and beyond it; then it was noted substrictural bronchus was much dilated, its walls lined with granulation tissue; dilated bronchus ended in a large cavity filled with mushy granulations (bronchiectasis and abscess); aspiration at various stages of the procedure removed a large quantity of foul pus; foreign body located in abscess cavity, freely movable in all directions.
Fbdy. 1247	4 yrs.	Brass headed tack	Right main bronchus, 12 days	None	5 mm. x 30 cm., standard	Point had penetrated bronchial wall	Tucker		Extraction Cure	4 min. 15 sec.	Oral	Point of tack projecting outward to right, through main bronchial wall.

Fbdy. 1250 1250	8 yrs.	Staple	Left main bronchus, 6 years	None	6 mm. x 35 cm., standard	Staple points upward. Points very sharp	Rotation	Curved end after version	Extraction Cure	18 min. 42 sec.	Oral	Foreign body history ignored. Patient treated for diphtheria, then for chronic bronchitis. Roentgen ray examination revealed foreign body. At bronchoscopy left bronchus quite inflammatory and blocked with granulations just above orifice of upper lobe bronchus; large quantities of pus came away in aspirator. Staple seized with rotation forceps and turned in an abscess cavity in left lower lobe, points rotating outward. Rotation method of removal.
Fbdy. 1258  1258	11 yrs.	Metallic object, portion automobile vibrator spring	Right lower lobe bronchus, 23 days	None	5 mm. x 40 cm., standard	Grasping a smooth, very hard steel object. Crosswise fixation requiring turning to avoid trauma.	Plain	Flatwise	Extraction Cure	31 min. 27 sec.	Oral	Spring found deep down in right lower lobe. Difficulty encountered due to position of thin, sharp-cornered spring end outward in a cavity it had made for itself by ulceration. Any traction in this position would have been dangerous. With the help of the roentgenologist's localization, foreign body was rotated into a safe position for removal with the end of the thin spring upward in tube mouth rendering possible removal without trauma.
Fbdy. 1265 1265	8 mos.	Tack	Left bronchus, 4 weeks	None	4 mm. x 20 cm., standard	Buried point	Tucker		Extraction Cure	9 min. 40 sec.	Oral	Roentgen ray because of cough and fever revealed unsuspected tack in bronchus, 3 weeks after probable aspiration. Tack found deep in left bronchus, head below orifice of left upper lobe bronchus.

HARDWARE—*(Continued)*

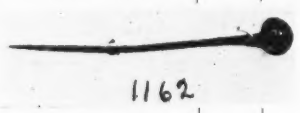
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1284—C-40	13 yrs.	Two brass headed thumb tacks	One in right stem bronchus, one in intestines, 24 hours	None	7 mm. x 40 cm., standard	None	Side grasping	Edge of head	Extraction Cure	14 min.	Oral	Tack found in right stem bronchus, with its upper margin in contact with spur of middle lobe bronchus. A small mass of thick secretion was adherent to shaft of tack. Point was not embedded. Edge of tack grasped and brought against tube mouth. It stripped off in trachea and became engaged on outside of bronchoscope. When bronchoscope was removed tack dropped into the pharynx and was swallowed. Tack recovered in stools two days later.
Fbdy. 1288	6 yrs.	Brass headed thumb tack	Right main bronchus, 2 days	None	6 mm. x 35 cm., standard	Guarding point	Plain	By point	Extraction Cure	2 min. 7 sec.	Oral	Thumb tack found in right main bronchus at orifice of upper lobe bronchus, point outward to right. Rotated and withdrawn with point protected in tube mouth. Thick tenacious pus coughed out through the bronchoscope.
Fbdy. 1381—C-95	3 yrs.	Pencil	Left main bronchus, 8 hours	None	5 mm. x 30 cm., standard	Tight impaction of pencil and edema proximal to it; no forcep spaces visible	Heavy forward grasping	Periphery	Extraction Cure	11 min. 30 sec.	Oral	Although aspirated but 8 hours before, the edematous swelling of the mucosa was so marked that the periphery of the pencil cap which was uppermost, could not be seen. There was marked obstructive atelectasis distal to the pencil.

Fbdy. 1383—C-97  C97-1383	8 yrs.	Screw	Left lower lobe bronchus, 1 month	None	6 mm. x 35 cm., aspirating	Head of screw upward. Difficulty in placing forceps blades at oposite poles on circumference of head of screw	Heavy forward grasping	Head of screw	Extraction Cure	24 min.	Oral	Prior to admission child was supposed to have an empyema (no knowledge of the presence of a screw). Needling attempted but no fluid had been obtained. Rib resection had been performed, no pus found. X-ray examination then revealed screw in right bronchus. Child extremely ill on admission. Roentgen ray showed screw in left lower lobe bronchus, head end uppermost. At bronchoscopy a great quantity of pus found in trachea coming from both main bronchi, greater quantity from right bronchus; granulation tissue found in left bronchus immediately below orifice of left upper lobe bronchus. With plain forceps, one jaw in fillester and one on side of head, screw was withdrawn to higher level; fresh grasp taken; foreign body stripped off at glottis; reaspirated into right stem bronchus from which it was removed. Many granulations found in right bronchus.
Fbdy. 1393 1393	23 yrs.	Staple	Left lower lobe bronchus about 2 cm. below left upper lobe bronchus, 15 years	Local	9 mm. x 45 cm., standard	Staple, two sharp points upward	Rotation	By points	Extraction Cure	17 min. 30 sec.	Oral	Staple completely buried in granulation tissue and hidden by masses of fibrous tissue on each side of walls making the lumen of stricture less in its transverse diameter than in the vertical. Bronchoscope insinuated into lumen of stricture and both points of staple brought into tube mouth. Thus sheathed staple was removed without trauma to bronchus. Points sheathed method of removal.

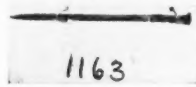
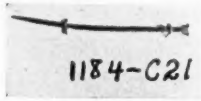
JEWELRY

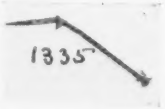
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1219	10 mos.	Collar button	Esophagus 6 weeks	None	5 mm. x 30 cm., standard esophagoscope. 4 mm. x 30 cm., standard bronchoscope for dyspnea	Extreme dyspnea			Foreign body passed on; recovered in stools Cure	31 min. 23 sec.		Child wheezy and dyspneic at times with harsh, crowing oral respiratory sounds. Bronchoscope passed; larynx and trachea intensely inflammatory; posterior tracheal wall bulging forward almost in contact with anterior wall on expiration; no foreign body in trachea; right and left bronchial orifices swollen and inflammatory. Esophagoscope passed with bronchoscope in situ; lumen swollen and filled with folds of edematous granulations. Ulceration at level indicated in ray (suprasternal notch); no foreign body seen. Tracheotomy necessary because of occlusion of trachea. Uneventful, but slow, recovery.
Fbdy. 1231	20 mos.	Collar button	Esophagus, level sixth cervical vertebra, 15 days	None	7 mm. x 30 cm., standard	Extreme dyspnea	Side-curved	Post	Extraction Cure	7 min. 35 sec.	Oral	Foreign body found deeply buried in lacerated and inflamamtory esophageal fold.

PINS AND NEEDLES

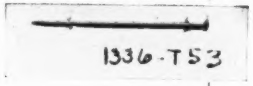
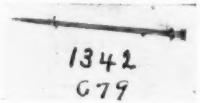
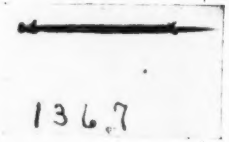
Fbdy. 1162 	18 yrs.	Shawl pin	Left bronchus head down, point near carina, 8 days	Local	8 mm. x 40 cm., aspirating	Shielding point	Tucker	Near point	Extraction Cure	1 min. 40 sec.	Oral	Pin originally in right bronchus, discovered in left bronchus at fluoroscopic examination just before bronchoscopy. Right main bronchus showed no pathology other than a reddening of mucosa. No pus and no appreciable degree of swelling. Point sheathed method with Tucker forceps.
Fbdy. 1246	3½ yrs.	Shawl pin	Internal branch of stem of left lower lobe bronchus about 7 weeks	None	6 mm. x 35 cm., standard		Plain	Point	Extraction Cure	14 min. 55 sec.	Oral	Pin discovered in bronchus, during Roentgen ray examination made because of bronchial symptoms. At bronchoscopy very thick, tough, gummy pus encountered in trachea and left main bronchus. This was aspirated. Fluoroscopic assistance of Dr. Manges.
Fbdy. 1252	18 yrs.	Shawl pin	Esophagus, just behind epiglottis, 6 days	None	Adult laryngoscope	Sheathing point; finding pin	Tucker	Near point	Extraction Cure	1 min. 5 sec.	Oral	About half shaft of pin buried in posterior wall of laryngopharynx. Point sheathed method with Tucker forceps.

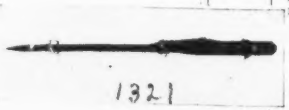
PINS AND NEEDLES—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1163 	51 yrs.	Common pin	Esophagus 6 days	None	Forbes speculum	Finding pin	Tucker	Head	Extraction Cure	2 min. 30 sec.	Oral	Pin found in anterior wall of the esophagus, the entire shaft of pin being buried up to head. Head of pin covered by mucosa.
Fbdy. 1184—C-21 	36 yrs.	Common pin	Pharynx, 2 days	None	Adult laryngoscope	Presentation and preventing escape downward	Alligator	Point	Extraction Cure	35 sec.	Oral	Pin found with head end extending forward in right pyriform sinus, point embedded in posterior pharyngeal wall, in midline. Patient placed in Jackson position.
Fbdy. 1217—T-31	17 yrs.	Common pin	Right pyriform sinus, few hours	None	Adult laryngoscope	Prevention of escape downward	Alligator	Pointed end	Extraction Cure	1 min. 10 sec.	Oral	Head of pin to right, point up. Patient placed in Jackson, low head position.
Fbdy. 1268—C-37	23 yrs.	Common pin with two small rubber spools attached	Right stem bronchus, 4 weeks	Local	9 mm. x 40 cm., standard	Point imbedded in internal wall. Foreign substance on shaft interfering with grasping of shaft	Tucker	Shaft near point	Extraction Cure	7 min. 25 sec.	Oral	Pin in right stem bronchus with point imbedded in inner bronchial wall on a level with orifice of middle lobe bronchus.

Fbdy. 1298 1298	12 yrs.	Common pin	Right lower lobe bronchus, below level of dome of diaphragm, 15 days	None	5 mm. x 30 cm., standard		Plain	Shaft near point	Extraction Cure	23 min. 52 sec.	Oral	Pin in a minute posterior branch of inferior lobe stem. Double plane fluoroscopic guidance by Dr. Manges.
Fbdy. 1312 1312	13 yrs.	Common pin	Left inferior lobe bronchus, 7 days	None	5 mm. x 45 cm., standard		Plain		Extraction Cure	10 min. 12 sec.	Oral	Pin found below level of dome of diaphragm in a small posterior branch of the left inferior lobe. Double plane fluoroscopic guidance by Dr. Manges.
Fbdy. 1314—C-49 1314-C49	17 yrs.	Common pin	Hypopharynx, 5 hours	None	Adult laryngoscope	Presentation and prevention of displacement downward	Tucker	Shaft near point	Extraction Cure	1 min. 56 sec.	Oral	Pin found in left side of hypopharynx imbedded posteriorly in pharyngeal wall and anteriorly at base of epiglottis. Patient placed in Jackson, head low position.
Fbdy. 1335  1335	21 yrs.	Common pin	Right pyriform sinus, 1 hour	Local	Adult laryngoscope	Transfixed in pyriform sinus. Presentation and disengaging of point	Alligator	By shaft	Extraction Cure	30 sec.	Oral	Point of pin which was bent, imbedded in wall of sinus. Patient placed in Jackson position.

PINS AND NEEDLES—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1336—T-53 	9 mos.	Common pin	Right stem bronchus, 1 week	None	4 mm. x 30 cm., standard	Presentation bronchoscope in line with pin	None, pin expelled through bronchoscope		Extraction Cure	1 min.	Oral	Larynx slightly inflammatory. 4 mm. tube fitted very closely in subglottic space but went through without undue force. A thread like line of pus found on posterior tracheal wall leading into right bronchus. Mucosa pale; otherwise normal. Bronchoscope was brought in line with right bronchus and advanced to level of upper lobe bronchus when child coughed violently and pin was projected through bronchoscope striking right lens of operator's glasses. Bronchoscope withdrawn with considerable resistance.
Fbdy. 1342—C-79 	4 yrs.	Common pin	Left main bronchus, 7 days	None	5 mm. x 30 cm., standard	Point buried	Tucker	Shaft near point	Extraction Cure	3 min. 15 sec.	Oral	Pin in left bronchus, head down, point imbedded in right tracheal wall. Small mass of granulation tissue at point of puncture of tracheal mucosa.
Fbdy. 1367 	6 yrs.	Common pin	Left lower lobe bronchus, anterior branch, 4 days	None	5 mm. x 30 cm., standard		Plain		Extraction Cure	8 min. 24 sec.	Oral	Double plane fluoroscopic guidance by Dr. Manges.

Fbdy. 1321 	9 yrs.	Sewing machine needle	Right inferior lobe bronchus, 4 days	None	7 mm. x 40 cm., standard	Finding	Plain	Near point	Extraction Cure	35 min. 15 sec.	Oral	Needle localized with the aid of Dr. Manges in a posterior branch of right inferior lobe bronchus.

SAFETY PINS

Fbdy. 1157	8 mos.	Open safety pin	Esophagus below cricopharyngeus, 1 day	None	Child's size laryngoscope	Open safety pin, point upward	Alligator	Near point	Extraction Cure	30 sec.	Oral	Point upward to left. Pin rotated to bring point into end of spatula where it was protected during removal.
Fbdy. 1160—C-16	9½ mo.	Open safety pin	Pharynx, 5 hours	None	Laryngoscope	Transverse position of pin	Alligator	Spring end	Extraction Cure	1 min. 25 sec.	Oral	Keeper to right lying behind right arytenoid, point downward and spring to left.

SAFETY PINS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1161	8 mos.	Open safety pin	Esophagus (point up), 6 days	None	5 mm., standard	Open safety pin, point up, obscured by and entangled with food	Tucker	Near point	Extraction Cure	9 min. 30 sec.	Oral	Large mass of food and fiber found above pin, and entangled with it, complicating the presentation.
Fbdy. 1164	14 mos.	Open safety pin	Larynx, 5 days	None	Laryngoscope	Open safety pin	Tucker (alligator form)	Near point	Extraction Cure	25 sec.	Oral	Open end up, point forward.
Fbdy. 1169	9 mos.	Open safety pin	Esophagus, cardiac end, 3 days	None	6 mm., full lumen	Open safety pin, point up; infant; dyspnea	Rotation	Spring end	Extraction Cure	14 min. 59 sec.	Oral	Point up. Removed by endogastric version

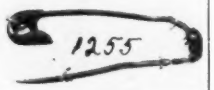
Fbdy. 1173 	9 mos.	Multiple foreign bodies (three safety pins, bent and fastened together in a burr with rubber band, in esophagus; also needle, pins, etc., in abdomen	Esophagus and alimentary tract, 7 days	None	Child's size laryngoscope	Burr-like mass of entangled and clamped safety pins with points in opposite directions	Rotation	By one point, other points protected by tube mouth	Extraction Cure	1 min. 55 sec.	Oral	Bunch of pins found entangled just below cricopharyngeus lying in bed of sloughing, ulcerated tissue. Odor of breath foul. Purulent secretion and whitish sloughs covered pins which were held together by a rubber band. Mass of pins rotated so that all points trailed when traction was made upward and all removed together. Child's condition quite septic. Temperature 102—103 prior to esophagoscopy. Needle lodged in intestines removed by abdominal surgeon. Child passed 2 buttons, one bent hair pin, two safety pins (closed), two bent common pins and several burnt match sticks and regurgitated other foreign bodies before admission. Shortly after admission passed additional match sticks, buttons and portions of hair pins. Child made a good recovery; discharged from hospital in excellent condition about two months after admission.
Fbdy. 1183 	11 mos.	Open safety pin	Stomach, 7 days	None	7 mm. x 45 cm., gastroscope	Open safety pin in pylorus of infant	Rotation	Ring	Extraction Cure	24 min. 55 sec.	Oral	Safety pin found lodged with keeper end in duodenum beyond the pylorus. Pointed branch was in stomach and could not go through. On inflation of stomach with air, pin did not drop free of pylorus, but required forceps removal. Pin turned and straightened in the tube and brought out along with tube. Per oral pyloroscopy. Double plane fluoroscopic guidance by Dr. Manges.

SAFETY PINS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1193—C 27	3 yrs.	Open safety pin	Esophagus, 7 days	None	Laryngoscope	Point imbedded	Rotation	Keeper end, point trailing	Extraction Cure	1 min. 15 sec.	Oral	Keeper of pin directed up and to right above cricopharyngeus. The point was imbedded in left wall of hypopharynx.
Fbdy. 1197	2 yrs.	Open safety pin	Esophagus, thoracic, 2 days	None	6 mm., standard	Open safety pin, point up	Rotation	Ring end	Extraction Cure	24 sec.	Oral	Point up in thoracic esophagus at crossing of left bronchus.

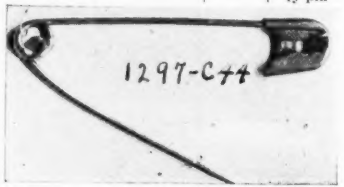
Fbdy. 1201—T 24	7 mos.	Open safety pin	Hypopharynx, 5 hours	None	Child's size laryngoscope	Point of seizure and traction to allow rotation and dangling	Rotation	Keeper end	Extraction Cure	45 sec.	Oral	Keeper end up and to left, point up, to right.
Fbdy. 1209—T 28	7 mos.	Open safety pin	Esophagus below cricopharyngeal fold, 24 hours	None	Esophageal speculum	Presentation. Avoidance of overriding and driving in of point	Alligator	Spring end	Extraction Cure	2 min. 25 sec.	Oral	Point down toward left; point imbedded in left lateral wall of esophagus.
Fbdy. 1216	9 mos.	Open safety pin	Esophagus just below cricopharyngeus, 21 hours	None	Child's size laryngoscope	Open safety pin	Tucker	Pointed branch	Extraction Cure	1 min.	Oral	
Fbdy. 1218—C-29	3 mos.	Open safety pin	Larynx, 36 hours	None	Child's size laryngoscope	Prevention of displacement downward	Alligator	Keeper	Extraction Cure	1 min. 23 sec.	Oral	Point of pin anteriorly imbedded in base of epiglottis, the keeper between arytenoids. Brought up dangling.

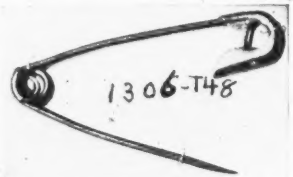
SAFETY PINS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1220	5 mos.	Open safety pin	Esophagus, 3 days	None	5 mm. gastroscope	Dyspnea; infant requiring small tube	Rotation		Extraction Cure	41 min. 22 sec.	Oral	Point up. Pin put into stomach, straightened out; gastroscope pushed down over pin; pin thus safely removed by per-oral gastroscopy.
Fbdy. 1255 	7 yrs.	Open safety pin	Right lower lobe bronchus, 1 month	None	5 mm., standard	Open safety pin in lung of child	Rotation and plain		Extraction Cure	18 min. 2 sec.	Oral	Pin found in stem of right lower lobe bronchus, both keeper and pin being below the orifice of the middle lobe bronchus; spurs between bronchial orifices thickened and inflammatory. Moderate bronchitis throughout right bronchi, but no sign of any trauma or exudate. Very little secretion and no pus. Pin grasped with rotation forceps and broken by traction against tube mouth into three portions; keeper branch withdrawn, leaving hook like pointed branch in bronchial stem; pointed branch removed with plain forceps; clamp on spring-end of pin dropped deeply into stem; removed with plain forceps. Dismembering method of removal. Fluoroscopic assistance by Dr. Manges.
Fbdy. 1260	21 mos.	Open safety pin	Esophagus, middle portion, 3 days	None	6 mm., standard and laryngoscope	Open safety pin, point up; thoracic esophagus; small child	Tucker	Pointed branch	Extraction Cure		Oral	Safety pin found in central portion of mediastinal esophagus, pointed branch extending strongly out to left. This branch seized with Tucker forceps, the branch rotated inward, then covered by sliding esophagoscope down over pointed branch of pin. Keeper of pin engaged in cricoid and required use of laryngoscope to disengaged it. Point sheathed method of removal.

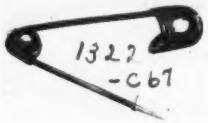
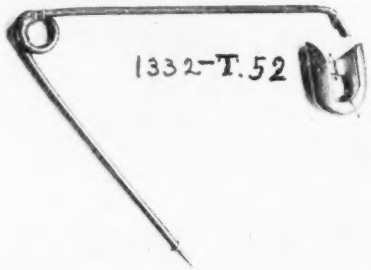
Fbdy. 1261	18 yrs.	Open safety pin	Deep down, left lower lobe, bronchus, 4 years	Local	5 mm., standard	Open safety pin deep in lung, secondary pathology from 4 years' sojourn	Side-curved	By pointed branch	Extraction Cure	28 min. 30 sec.	Oral	Left main bronchus collapsed in its upper portion; lower lobe bronchus not collapsed but occluded with fibrous tissue and granulations. Main portion of pin found deep down in left lower lobe. Pointed branch brought into tube and was withdrawn. Keeper located at a still deeper level and was too large to come out through bronchoscope. The pin was very friable from corrosion. Pin discovered by an X ray for a supposed pneumonia 4 years after foreign body history had been forgotten.
Fbdy. 1272	4 mos.	Open safety pin	Esophagus below level of upper border of sternum, 3 days	None	5 mm., standard	Infant; avoidance of dyspnea during manipulations	Tucker	Pointed branch	Extraction Cure	10 min. 15 sec.	Oral	Removed by point-sheathed method.
Fbdy. 1273	7 mos.	Open safety pin with mass of wool attached	Esophagus just above supra sternal notch, 5 days	None	5 mm., standard	Infant. Pin entangled with wool. Point of pin in mediastinum	Tucker		Portion of wool extracted. Safety pin advanced into stomach. Death	20 min. 35 sec.		Patient died of mediastinal complications.

SAFETY PINS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1282	1 yr.	Open safety pin	Esophagus, lower end, 6 days	None	6 mm. trap-door esophagoscope	Open pin deep in esophagus of infant	Tucker	Pointed branch	Extraction Cure	6 min. 30 sec.	Oral	Safety pin found in lower end of esophagus with spring end downward engaged in hiatus esophageus. Pointed branch which was upward toward right, seized with Tucker forceps; trap-door esophagoscope pushed downward until pointed branch rested against the end of tube-mouth. Forceps rotated and point dropped into distal end of esophagoscope. Esophagoscope, forceps and pin withdrawn, keeper end outside.
Fbdy. 1291	11 mos.	Open safety pin	Esophagus, about 2 cm. above diaphragm, 4 days	None	6 mm. trap-door esophagoscope	Open safety pin; point up; thoracic esophagus; infant	Tucker	Point	Extraction Cure	4 min. 5 sec.	Oral	Small tube required because of size of infant.
Fbdy. 1297—C-44 	18 mos.	Open safety pin	Pharynx 2 hrs.	None	Child's size laryngoscope	Very large pin with stiff spring	Alligator	Pointed shaft	Extraction Cure	3 min. 15 sec.	Oral	Spring end downward on level with epiglottis, keeper and point being behind soft palate. Pointed shaft grasped and carried obliquely downward; brought forward from behind soft palate.

Fbdy. 1306—T-48 	2 yrs.	Open safety pin	Hypopharynx and esophagus, 1 day	None	Child's size laryngoscope	Point sheathed by end of laryngoscope	Alligator	Keeper	Extraction Cure	1 min. 47 sec.	Oral	Child developed a laryngotracheitis the day before the pin lodged. At the end of three days, infection cleared up.
Fbdy. 1310—T-49	6 mos.	Open safety pin	Hypopharynx, 10 hours	None	Child's size laryngoscope	Dyspneic infant	Tucker	Keeper. Pin rotated; removed from pharynx by grasping spring	Extraction Cure	5 min. 2 sec.	Oral	Point and keeper upward and posterior to larynx, spring below cricopharyngeal fold. Point imbedded in left pyriform sinus. Oxygen administered during removal.
Fbdy. 1311	5 mos.	Open safety pin	Esophagus, upper third, 2 days	None	5 mm., standard	Infant 5 months old with mediastinitis on admission	Tucker	Pointed branch	Extraction Death	18 min. 45 sec.	Oral	Mother attempted removal with finger. At esophagoscopy, esophagus found swollen shut with bluish-white edematous folds entirely obscuring the pin. There were old blood clots adherent to esophageal wall. When these came away in the aspirator, the spring end of pin was revealed; both keeper and pointed branches being concealed in the tissue of the esophageal wall. Pin moved downward to disengage buried branches of pin. Point brought into esophagoscope by pushing esophagoscope down over point; pin then removed. Died of mediastinal complications.

SAFETY PINS—*Continued*

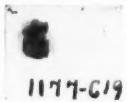
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1322—C-67 	10 mos.	Open safety pin	Hypopharynx, 3 days	None	Child's size laryngoscope	Point up	Alligator	Keeper	Extraction Cure	45 sec.	Oral	Keeper directed to left with point to right and spring end down behind cricoid.
Fbdy. 1332—T-52 	36 yrs.	Open safety pin	Esophagus and hypopharynx, 1 day	None	Adult laryngoscope	Very large stiff pin	Alligator rotation	Keeper and spring	Extraction Cure	7 min. 55 sec.	Oral	Pin found in hypopharynx, keeper and point up with spring in the esophagus below cricopharyngeal fold. Point and shaft were to right imbedded in right lateral pharyngeal wall. Point retreated over end of laryngoscope. Keeper pulled off; spring grasped with rotation forceps; pin rotated. Patient was an inmate of a hospital for mental diseases and swallowed foreign bodies voluntarily. X-ray revealed a second safety pin, a needle and a common pin in upper abdomen. Intestinal safety pin recovered in stools. Needle and pin removed by laparotomy from transverse colon when it was found they showed no movement onward. Uneventful recovery. Patient discharged well.

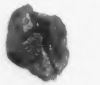
Fbdy. 1368—T-69	2 yrs.	Open safety pin	Esophagus, level suprasternal notch, 1 day	None	7 mm., standard	Open safety pin in thoracic esophagus	Rotation	Keeper	Extraction Cure	1 min. 3 sec.	Oral	Point of pin up; angle of opening, 45 degrees, Keeper to left; point to right in thoracic esophagus. Removed point trailing, after rotating.
Fbdy. 1370	15 mos.	Open safety pin	Esophagus, 2 cm. below cricopharyngeus, 2 days	None	6 mm., standard	Open safety pin in 15 months old child	Rotation	Keeper	Extraction Cure	1 min. 48 sec.	Oral	Dangling method of removal.
Fbdy. 1378—C-93	11 mos.	Open safety pin	Esophagus below cricopharyngeus, 48 hours	None	Child's size anterior commissure laryngoscope	Prevention of overriding. Point downward and deeply imbedded	Rotation	Spring	Extraction Cure	50 sec.	Oral	Spring end of pin uppermost and directed toward right on level with right arytenoid; pointed shaft directed to left and downward, the point imbedded. Keeper below cricopharyngeus.

SAFETY PINS—*Continued*

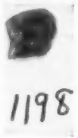
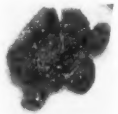
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1398—T-79	7 mos.	Open safety pin	Esophagus and hypopharynx, 1 day	None	Child's size laryngoscope	Rotation to allow point to trail	Rotation	By keeper	Extraction Cure	6 sec.	Oral	Safety pin found in hypopharynx and esophagus; keeper in left pyriform sinus, point in right pyriform sinus, spring below cricopharyngeal fold; pin lying in lateral plane posterior to cricoid cartilage, point up.

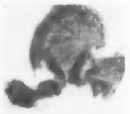
SEEDS, NUTS AND SHELLS

Fbdy. 1177—C-19 	5 yrs.	Bean	Right main bronchus, 2 days	None	5 mm. x 30 cm., standard	Swollen mucosa, large quantity of secretion	Side-curved	Minor axis	Extraction Cure	8 min. 17 sec.	Oral	Marked subglottic swelling present; tracheal mucosa intensely red and swollen. Considerable quantity of whitish secretion aspirated. Small portion of material apparently skin of bean found in left bronchus. Tracheotomy required for dyspnea. Before bronchoscopy signs of a shifting foreign body loose in trachea were present.
Fbdy. 1178	2½ yrs.	Peanut kernel	Left inferior lobe bronchus below orifice of upper lobe, 16 days	None	5 mm. x 30 cm., standard	Vegetal bronchitis; friable fbdy.	Side-curved		Extraction Cure	7 min. 30 sec.	Oral	Orifice of left bronchus just below bifurcation almost completely occluded by granulation tissue. Secretions aspirated prior to finding foreign body.

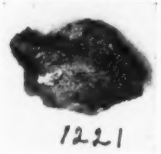
Fbdy. 1179	26 mos.	Peanut kernel	Right bronchus, 20 days	None	5 mm. x 30 cm., standard	Vegetal bronchitis; friable fbdy.; impacted; no forceps spaces	Side-curved		Extraction Cure	5 min. 20 sec.	Oral	Foreign body wedged in right bronchus. Lost in mouth during removal. Secretions aspirated.
Fbdy. 1182	17 mos.	Peanut kernel	Right bronchus at orifice of upper lobe, 17 days	None	5 mm. x 30 cm., standard	Vegetal bronchitis; friable fbdy. Proximal edema; infant	Side-curved		Extraction Cure	1 min. 45 sec.	Oral	Large quantity of purulent secretion removed with aspirator on introduction of bronchoscope.
Fbdy. 1189—C-25 	5 yrs.	Peanut kernel	Right main bronchus, 6 days	None	6 mm. x 35 cm standard	Large piece of peanut; swollen mucosa; forceps spaces small and only on inspiration	Peanut	Minor axis	Extraction Cure	25 min. 30 sec.	Oral	Marked laryngotracheobronchitis with considerable amount of secretion, removed with aspirator before foreign body was visible.
Fbdy. 1190	2 yrs.	Peanut kernel	Trachea, 2 mos.	None	5 mm. x 30 cm., standard	Pathology; vegetal bronchitis; friable fbdy.	Side-curved		Extraction Cure	1 min. 50 sec.	Oral	Subglottic tissue very much swollen. Trachea intensely inflammatory. Pus abundant. Foreign body loose in trachea at time of removal. Pus aspirated.
Fbdy. 1196	2½ yrs.	Watermelon seed	Loose in trachea at bifurcation. 5 days	None	5 mm. x 30 cm. standard	Swollen mucosa and pus; bad condition of patient.	Side-curved	Flatwise	Extraction Death	54 sec.	Oral	Intense laryngotracheobronchitis; subglottic tissue swollen almost shut. In ward few hours later patient stopped breathing. Emergency tracheotomy done by interne but patient failed to respond to efforts to restore breathing.

SEEDS, NUTS AND SHELLS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1198 	2 yrs.	Peanut kernel	Right main bronchus, below level of middle lobe bronchus, 9 days	None	4 mm. x 30 cm., standard	Pathology; vegetal bronchitis; acute proximal stenosis; friable fbdy.	Side-curved		Extraction Cure	1 min. 10 sec.	Oral	Subglottic tissue very much swollen; tracheobronchial mucosa swollen; right bronchus swollen, nearly shut below level of middle lobe bronchus. Peanut kernel found below this swelling. Pus aspirated before foreign body seen.
Fbdy. 1204 	18 mos.	Peanut kernel	Trachea, right and left bronchi, 24 hours	None	5 mm. x 30 cm., standard	Multiple foreign bodies; impending asphyxia friable fbdy; infant;	Side-curved	Minor axis	Extraction Cure		Oral	Portion of peanut kernel found in right lower lobe bronchus, at orifice of middle lobe bronchus. Removed. Deemed best to delay further bronchoscopy for further localization of other fragments. Child brought back to operating room with breathing arrested; color deeply cyanotic. Bronchoscope quickly introduced. A large portion of peanut kernel located at bifurcation occluding both bronchi; removed with side-curved forceps. Breathing not being clear, bronchoscope reintroduced, pus aspirated; half peanut kernel found in left bronchus and removed. Because of extensive edema and intense bronchitis, tracheotomy became necessary 36 hours after bronchoscopy. Child made a good recovery. Father (a physician) present at bronchoscopy expressed appreciation of team work and also of work without anesthesia, which in this case would have been fatal because of the respiratory arrest.

Fbdy. 1205 1205	4 yrs.	Watermelon seed	Left bronchus, 8 days	None	4 mm. x 30 cm., standard	Inflammatory condition of mucosa (vegetal bronchitis)	Side-curved	Flatwise	Extraction Cure	2 min. 25 sec.	Oral	Intense, diffuse tracheobronchitis.
Fbdy. 1206  1206	3 yrs.	Dried pea	Right main bronchus, 5 days	None	5 mm. x 30 cm., standard	Swollen, soft friable foreign body	Side-curved		Extraction Cure	6 min. 35 sec.	Oral	Vegetal bronchitis; secretions aspirated before foreign body could be seen.
Fbdy. 1207	3 yrs.	Grain of corn (maize)	In trachea at bifurcation, 10 days	None	4 mm. x 30 cm., standard	Mucosal swelling; vegetal bronchitis	Side-curved		Extraction Cure	38 sec.	Oral	Subglottic tissue swollen until the two sides nearly touched in the midline on inspiration. Laryngeal and tracheal mucosa intensely inflammatory. Grain of corn found at bifurcation of trachea crosswise occluding both bronchi. Pus aspirated. Tracheotomy for dyspnea required 15 minutes after bronchoscopy. Decannulated after 8 days.
Fbdy. 1208—T 27 1208–T27	2 yrs.	Grain of corn (maize)	Trachea and left bronchus, 3 days	None	4 mm. x 30 cm., standard.	Grasping of moving foreign body; vegetal bronchitis	Side-curved	Germ end	Extraction Cure	2 min. 20 sec.	Oral	Pus in pharynx and coming up through larynx. Violent tracheobronchitis with large quantities of purulent secretion but not the usual amount of subglottic swelling. Foreign body swollen.

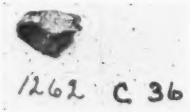
SEEDS, NUTS AND SHELLS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1214	16 mos.	Watermelon seed	Trachea, 9 days	None	4 mm. x 30 cm., standard	Moving slippery foreign body. Vegetal bronchitis	Side-curved	Flatwise	Extraction Cure	30 sec.	Oral	Subglottic tissue intensely swollen so that even a 4 mm. bronchoscope was a very tight fit. Diffuse tracheobronchitis. Foreign body loose in trachea. Tracheotomy necessary 3 hours after bronchoscopy. Decannulated 9 days later.
Fbdy. 1215	3 yrs.	Pecan nut kernel	Left bronchus, 10 days	None	5 mm. x 30 cm., standard	Small multiple foreign bodies; friable	Side-curved		Extraction Cure	17 min. 35 sec.	Oral	Right bronchus contained small crumbs of whitish material (nut kernel) which was not obstructive. Left bronchus found completely obstructed at orifice of left upper lobe bronchus. A number of portions of nut kernel removed. Left upper lobe bronchial orifice very much swollen and secretions seen emerging from it. Purulent secretion and crumbs aspirated.
Fbdy. 1221 	60 yrs.	Portion of ginger root	Left main bronchus, 4 days	Local	8 mm. x 40 cm., standard	Tight impaction; proximal edema	Plain	Over minor axis	Extraction Cure	1 min. 36 sec.	Oral	Foreign body completely obstructing left main bronchus; foreign body nonopaque but X-ray diagnosis positive, as in all peanut cases.
Fbdy. 1225	4 yrs.	Peanut kernel	Trachea at bifurcation, 4 days	None	5 mm. x 30 cm., standard	Dyspnea; friable foreign body	Side-curved	Minor axis	Extraction Cure	45 sec.	Oral	Nut kernel found at bifurcation transfixed crosswise so as partially to occlude the orifices of both main bronchi.

Fbdy. 1234 	2 yrs.	Peanut kernel	Right bronchus, 2 days	None	5 mm. x 30 cm., standard	Soft friable foreign body; swollen mucosa	Side-curved		Extraction Cure	1 min. 31 sec.	Oral	Subglottic tissues very much swollen; diffuse laryngotracheobronchitis. Secretions aspirated before foreign body found.
Fbdy. 1239 	9 yrs.	Prune pit	Right main bronchus, orifice upper lobe, 7 days	None	6 mm. x 35 cm., aspirating	Tight impaction; forceps spaces small and only on inspiration	Plain	Minor axis	Extraction Cure	1 min. 10 sec.	Oral	Foreign body covered with pus; forceps spaces exceedingly small and occluded completely at beginning of expiration. Purulent secretions aspirated before foreign body seen. Positive Roentgen ray diagnosis, nonopaque foreign body, by Manges, as in all cases of nut kernel, maize, etc., by obstructive atelectasis and obstructive emphysema.
Fbdy. 1240—C-32 	6 yrs.	Grain of corn (maize)	Right main bronchus, 4 days	None	6 mm. x 35 cm., standard	None	Forward grasping	Flatwise	Extraction Cure	12 min. 40 sec.	Oral	Moderate swelling of subglottic tissues and general redness of mucous membrane of trachea.

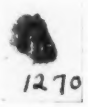
SEEDS, NUTS AND SHELLS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1245—T-35	3 yrs.	Peanut kernel	Right lower lobe bronchus, 11 days	None	5 mm. x 30 cm., standard	Presentation; no forceps spaces; fbdy. in external branch right lower lobe bronchus; acute proximal edema; friable fbdy.	Side-curved	Minor axis	Extraction Cure	3 min. 35 sec. 3 min. 25 sec.	Oral	*First Bronchoscopy*: Larynx and trachea inflammatory, moderate degree of subglottic swelling. Free pus in trachea. Pus coming from right bronchus, but bronchial orifice devoid of pus after coughing. Right bronchial mucosa was inflammatory. Pus aspirated. Small portion of peanut removed from small external branch, right lower lobe bronchus. Mucosa of lower lobe bronchi swollen and granular. Presence of other particles of peanut not excluded. Further exploration deferred until indicated by physical signs and additional ray studies. *Second Bronchoscopy* (2 weeks later): Free pus coming up through larynx. Tracheal mucosa pale with little swelling. Pus aspirated from trachea. After coughing the left bronchus was clear of pus; very slightly inflammatory. Right bronchus filled with thick yellowish odorless pus. Mucosa granular and swollen. Pus aspirated and lower bronchi explored. Granulations in lower lobe bronchi cleared with swab and pus aspirated. No additional fragments of foreign body found. Slow but perfect recovery from vegetal bronchitis.

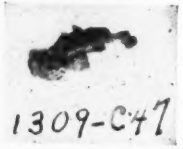
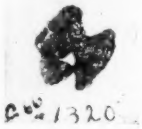
Fbdy. 1251 1251	3 yrs.	Peanut kernel	Right bronchus, 1 month (removed from left bronchus)	None	5 mm. x 30 cm., standard	Extreme dyspnea; fbdy. aspirated into sound lung; other functionless from long sojourn of fbdy. Friable fbdy.	Side-curved	Minor axis	Extraction Cure	9 min. 15 sec.	Oral	Upon insertion of bronchoscope, patient became very cyanotic. Right bronchus found intensely inflammatory, the walls lined with granulation tissue, and large quantities of pus came up through aspirator. Left bronchus somewhat less inflammatory. Peanut kernel found in left bronchus at orifice of left upper lobe bronchus, and removed. It is probable that the peanut kernel shifted on introduction of bronchoscope which accounted for cyanosis. The site of sojourn of the peanut kernel in right bronchus was very plainly indicated by granulations, subjacent pus and thick, tenacious secretions lining wall of bronchus. These were aspirated.
Fbdy. 1262—C 36  1262 C 36	19 yrs.	Seed	Right lower lobe bronchus, 7 weeks	Local	8 mm. x 40 cm., standard	Obscured by granulations	Side-curved	Minor axis	Extraction Cure	3 min. 24 sec.	Oral	Small mass of granulation tissue seen at each side of foreign body.
Fbdy. 1263 1263	1½ yrs.	Peanut kernel	Left main bronchus, 17 days	None	4 mm., standard	Proximal acute inflammatory stenosis; tight impaction from prolonged sojourn; friable fbdy.	Side-curved		Extraction Cure	2 min. 8 sec.	Oral	Intense subglottic swelling; cords much swollen; tracheal mucosa very inflammatory and covered with thick secretion; left bronchial orifice intensely inflamed; right bronchial orifice less so. Peanut kernel found tightly clasped in inflammatory tissue at the widening in left main bronchus corresponding to orifice of upper and lower lobe bronchi. Only air space visible past it was on flat side of peanut kernel; slight bubbling of secretion past this space.

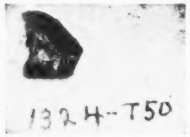
SEEDS, NUTS AND SHELLS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1266—T-38	18 mos.	Peanut kernel	Right upper lobe bronchus, 5 days	None	4 mm. x 30 cm., standard	Forceps spaces only on inspiration and small; friable fbdy.	Side-curved	Equator	Extraction Cure	2 min. 45 sec.	Oral	Larynx inflammatory; tracheal mucosa slightly edematous; trachea contained considerable pus, yellowish in color. Orifice of left bronchus inflammatory and slightly edematous. Purulent secretions aspirated during bronchoscopy.
Fbdy. 1269	2 yrs.	Bean	Left main bronchus, 3 weeks	None	4 mm., standard	Pathology from 3 weeks' sojourn (vegetal bronchitis); moribund condition of patient	Side-curved	Minor axis	Extraction Death	10 min. 40 sec.	Oral	Child quite ill on admission. At bronchoscopy a quantity of thick pus was found adherent to walls of trachea; subglottic tissue swollen; right bronchial orifice seemingly larger than normal; left bronchial orifice closed to a scabbard-shaped lumen containing a pool of pus through which no air was passing in or out. Aspiration of pus revealed a lumen just below the orifice swollen entirely shut, being collapsed from before backward. Aspirator inserted through this bringing up large quantities of pus, very foul. Edge of bean then visible buried in swollen mucosa; removed. Oxygen administered through bronchoscope throughout entire bronchoscopy. Following bronchoscopy patient failed to improve. Coughed up large quantities of foul pus. Respiration difficult. Death occurred about 36 hours after bronchoscopy, from continuation of diffuse suppurative pneumonitis

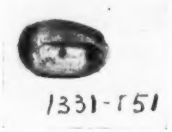
Fbdy. 1270 	26 mos.	Peanut kernel	Right bronchus, 3 weeks	None	5 mm. x 30 cm., standard	Acute proximal inflammatory stenosis; friable fbdy.	Side-curved	Minor axis	Extraction Cure	9 min. 30 sec.	Oral	Peanut kernel impacted in granulation tissue in right bronchus. Subglottic tissue very much swollen. Mucosa of all bronchi very much swollen. Portions of peanut kernel removed; another portion coughed out through bronchoscope. Tracheotomy necessary for dyspnea 3 days after bronchoscopy. Ultimate recovery.
Fbdy. 1277—T-42	35 yrs.	Peanut kernel	Esophagus, 1 day	None	7 mm. x 30 cm., standard	Impacted in lumen of stricture	Forward grasping	Minor axis	Extraction Cure		Oral	History of swallowing lye in childhood; difficulty in swallowing since. At esophagoscopy esophageal lumen found to be narrowed to about 3 mm. diameter at the level of the cricopharyngeal fold. Peanut kernel completely blocking lumen. Constriction apparently due to cicatricial tissue.
Fbdy. 1281	50 yrs.	Orange seeds	Esophagus in organic narrowing, 1 day	None	9 mm. x 40 cm., standard	Impaction in lumen of stricture	Forward grasping (hollow faced)	Minor axis	Extraction Cure	4 min. 50 sec.	Oral	Patient had a firm infiltrative lesion of the esophagus with narrowing at hiatus esophageus so small as to prevent the passage downward of an orange seed.
Fbdy. 1283	16 mos.	Peanut kernel	Right stem bronchus, 7 weeks	None	4 mm. x 30 cm., standard	Proximal swollen mucosa; 7 weeks' inflammatory; friable fdby; infant	Side-curved	Minor axis	Extraction Cure	6 min. 32 sec.	Oral	Subglottic tissue swollen; trachea intensely inflamed. Left bronchial orifice also inflamed; right main bronchus swollen so as to diminish its lumen to about one-half normal. Peanut kernel found in right stem bronchus just below orifice of upper lobe bronchus, surrounded by granulation tissue. Pus aspirated during bronchoscopy.

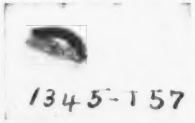
SEEDS, NUTS AND SHELLS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1292	25 mos.	Chestnut hull	Left bronchus at orifice of left upper lobe, 3 days	None	5 mm. x 30 cm., standard	Swollen mucosa	Side-curved	Flatwise	Extraction Cure	11 min. 50 sec.	Oral	Asthmatoid wheeze noticed very markedly just before bronchoscopy. This is especially interesting in view of the depth at which foreign body was located. Pus aspirated during bronchoscopy. Characteristic vegetal bronchitis from chestnut hull.
Fbdy. 1295—C-43	11 mos.	Peanut kernel	Trachea, one end in left bronchus, 1 day	None	4 mm. x 30 cm., standard	Very large portion of peanut in infant; dyspnea; friable fbdy.	Side-curved	Minor axis	Extraction Cure	2 min. 10 sec.	Oral	Large peanut found in lower end of trachea with one end in left bronchus, the other end extending across the carina towards the orifice of the right main bronchus. Mucosa of the lower trachea and orifices of both main bronchi inflammatory. Pus aspirated during bronchoscopy.
Fbdy. 1309—C-47 	2 yrs.	Peanut	Right main bronchus, 3 days	None	5 mm. x 30 cm., standard	Swollen mucosa with purulent secretion. No forceps spaces until obtained by manipulation; proximal annular edema; friable fbdy.	Side-curved		Extraction Cure	8 min. 17 sec.	Oral	Larynx and trachea inflammatory. Pus found in right bronchus; removed with aspirator. Peanut was seen in right main bronchus partially obscured by a ring of swollen mucosa and secretion. Secretions aspirated during bronchoscopy.
Fbdy. 1320—C-66 	2 yrs.	Peanut kernel	Right main bronchus, 1 week	None	5 mm. x 30 cm., standard	Large peanut; marked proximal swelling of mucosa, large quantity of secretion; forceps spaces only on deep inspiration; friable fbdy.	Side-curved	Minor axis	Extraction Cure	4 min. 30 sec.	Oral	Mucosa of trachea inflammatory. A large peanut kernel found in orifice of bright main bronchus. Considerable secretion present which was removed with independent aspirating tube.

Fbdy. 1324 	16 yrs.	Peanut kernel	Right lower lobe bronchus, 6 days	None	6 mm. x 35 cm., standard	Presentation in pus and swollen mucosa; proximal annular edema; friable fbdy.	Side-curved	Minor axis	Extraction Cure	6 min. 5 sec.	Oral	Free pus found in trachea, found to be coming from right main bronchus. Tracheal and bronchial mucosa very inflammatory. No subglottic swelling noted. Foreign body found covered with pus and edematous mucosa. Large quantity of pus aspirated during bronchoscopy.
Fbdy. 1327	18 mos.	Peanut kernel	Right bronchus at orifice of middle lobe, 11 days	None	4 mm. x 30 cm., standard	Friable fbdy. Proximal annular edema; infant	Side-curved	Minor axis	Extraction Cure	1 min.	Oral	Intense laryngotracheobronchitis; left bronchial orifice very inflammatory; bronchus not explored. Right bronchus intensely inflamed. Peanut kernel found wedged at orifice of middle lobe bronchus; right upper lobe bronchial orifice swollen nearly shut. Secretions aspirated.
Fbdy. 1328	9 mos.	Portion flower stem	Right bronchus at orifice of middle lobe, 4 days	None	4 mm. x 30 cm., standard	Size of patient (infant). Proximal annular edema	Side-curved	Minor axis	Extraction Cure	3 min. 20 min.	Oral	Bronchial and tracheal mucosa swollen. Subglottic tissue very much swollen so that the 4 mm. bronchoscope was a close fit. Left bronchus swollen at orifice but not explored. Right bronchus swollen nearly shut. Pus aspirated during bronchoscopy. Typical vegetal bronchitis.
Fbdy. 1330—C-72	22 mos.	Peanut kernel	Left lower lobe bronchus, 5 days	None	5 mm. x 30 cm., standard	Intense inflammatory reaction, ring of edema almost completely obscuring foreign body; friable fbdy.	Side-curved	Flatwise	Extraction Cure	13 min. 20 sec.	Oral	Mucosa of trachea and bronchi very inflammatory, with swelling of the subglottic tissues. Considerable quantity of secretion aspirated from left side. Peanut kernel seen in left lower lobe bronchus immediately below orifice of left upper lobe bronchus, almost completely hidden by ring of edema. Tracheotomy for dyspnea necessary 24 hours after bronchoscopy. Decannulated 3 weeks later.

SEEDS, NUTS AND SHELLS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1331—T-51 	3 yrs.	Grain of corn (maize)	Trachea and left main bronchus, 3 days	None	5 mm. x 30 cm., standard	Foreign body swollen to three times normal size. Dyspnea	Forward grasping	Large end	Extraction Cure	4 min. 20 sec.	Oral	Slight subglottic swelling. Moderate degree of tracheitis. Mucopus coming up freely from both right and left bronchi. Grain of corn found at bifurcation with small end in left bronchus. Secretions aspirated during bronchoscopy. Vegetal bronchitis from maize.
Fbdy. 1334—C-74	33 yrs.	Peanut kernel	Right lower lobe bronchus, 4 weeks	Local	8 mm. x 40 cm., standard	Granulation and secretion obscuring foreign body; friable fbdy.	Plain	Minor axis	Extraction Cure	7 min. 55 sec.	Oral	There was considerable secretion seen coming from right bronchus. This was aspirated. Mucosa of trachea and right bronchus slightly inflammatory. Secretion seen to be coming from right lower lobe bronchus, the orifice of which was occluded by the foreign body and granulation tissue. On inspiration forceps spaces could be seen laterally. Peanut removed through bronchoscope. After removal of foreign body a large quantity of secretion was aspirated from beyond point of lodgment of foreign body, and a large granulation was removed. Inflammatory condition mild as compared to that seen in children.

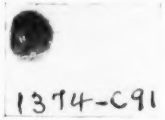
Fbdy. 1339—T-54	6 yrs.	Grain of corn (maize)	Trachea and left main bronchus, 13 days	None	5 mm. x 30 cm., standard	Presentation of moving foreign body	Forward grasping	Flatwise, well down over germ end	Extraction Cure	1 min. 44 sec.	Oral	Larynx and trachea found intensely inflamed; free pus in trachea coming up from below. On introduction of bronchoscope the foreign body was coughed against end of the tube. As the bronchoscope was introduced further the foreign body retreated ahead of bronchoscope, going down left bronchus, whence it was removed. The tracheobronchial mucosa intensely inflammatory and edematous with patches of erosion and considerable pus throughout tracheobronchial tree.
Fbdy. 1341—C-78	4½ yrs.	Watermelon seed	Right main bronchus, 7 days	None	5 mm. x 30 cm., standard	None	Side-curved	Flatwise	Extraction Cure	1 min. 20 sec.	Oral	Moderate subglottic swelling. Mucosa of entire tracheobronchial tree inflammatory. Secretions aspirated.
Fbdy. 1345—T-57 	2½ yrs.	Peanut kernel	Right main bronchus, 4 days	None	4 mm. x 30 cm., standard	Presentation and forceps spaces. Friability of fbdy.	Forward grasping, stiff spring	Minor axis; end	Extraction Cure	4 min. 21 sec.	Oral	Slight swelling of the subglottic tissue. Slight inflammatory reaction in the trachea with small amount of mucopus. Peanut found in right main bronchus at orifice of upper lobe br. Mucosa of right main and right upper lobe bronchus very inflammatory. Mucopus seen coming up from around peanut. Pus aspirated during bronchoscopy.

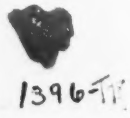
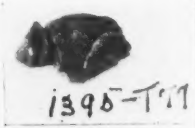
SEEDS, NUTS AND SHELLS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1351—T-61	2 yrs.	Peanut kernel	Right lower lobe bronchus, 14 days	None	4 mm. x 30 cm., standard	Presentation in pus and granular mucosa; very ill child; friable fbdy.	Forward grasping	Minor axis	Extraction Cure	15 min. 5 sec.	Oral	On admission child very ill. Dyspnea was so urgent that tracheotomy was necessary. Peroral bronchoscopy (five days later): Tracheobronchial mucosa inflammatory with free pus coming principally from right lower lobe bronchus. After aspiration, foreign body found in an external division of right lower lobe bronchus distal to a ring of edematus mucosa and was only visible on deep inspiration. Uneventful recovery with decannulation.
Fbdy. 1358—C-85	3 yrs.	Peanut kernel	Right main bronchus, 7 days	None	6 mm. x 35 cm., standard	None, except friability of fbdy.	Side-grasping, stiff spring	Flatwise	Extraction Cure	3 min. 2 sec.	Oral	Mucosa of trachea and both main bronchi moderately inflammatory. A few small granulations were seen in left main bronchus. It is very probable from the endobronchial findings that the foreign body had at one time been in the left main bronchus and had recently shifted to the right side. Secretions aspirated during bronchoscopy.

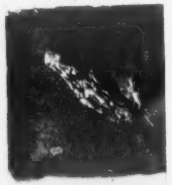
Fbdy. 1360—T 65	19 mos.	Peanut kernel	Trachea, at bifurcation, 3 days	None	4 mm. x 30 cm., standard	Presentation. Proximal edema; infant	Side-grasping	Minor axis beyond presenting end	Extraction Cure	3 min. 54 sec.	Oral	Mucous membrane of larynx and trachea showed a great deal of inflammatory reaction with a thin mucopus adherent. There was very little subglottic swelling. Foreign body seen at level of carina. Found to be cone-shaped, apex upward, the base quadrilateral in shape. The position of the foreign body at bifurcation was such that about one-half of lumen of right bronchus could be seen; the left bronchial orifice was closed to about 1/6 of normal lumen. The peanut showed a fresh break and it was thought advisable to re-insert bronchoscope, but no additional portions of peanut seen. The left bronchus showed a ring of edema about half way between carina and left upper lobe bronchus; the lumen of this narrowing was half that of bronchus. Secretions aspirated.
Fbdy. 1363—C 88	22 mos.	Peanut kernel	Right main bronchus, 3 days	None	4 mm. x 30 cm., standard	None, except friability of fbdy.	Side-curved	Equator	Extraction Cure	3 min. 1 sec.	Oral	Moderate redness of larynx and trachea. Small amount of thin secretion present; aspirated.

SEEDS, NUTS AND SHELLS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Route	Time	Comment
Fbdy. 1371—T-71	2 yrs.	Bean	Right main bronchus, 2 days	None	4 mm. x 30 cm., standard	Tight impaction of swollen bean; friable fbdy.	Forward grasping deeply cupped	Presenting end and minor axis of fragments	Extraction Cure	20 min. 28 sec.	Oral	Trachea showed marked deviation toward right at level of suprasternal notch. Foreign body tightly wedged in main bronchus, there being no evidence of forceps spaces on deep inspiration. Because of large size of bean and the possibility of its being stripped off at the glottis on withdrawal, and the strong probability of re-aspiration into the left side, and the fact that the entire right lung was temporarily out of function, it was determined to remove the bean in fragments. Bronchoscope reinserted after all portions of bean removed, and right bronchus aspirated free of pus. Tracheotomy for dyspnea necessary about 24 hours after bronchoscopy. Decannulated about 3 weeks later. Discharged well. Typical atalectasis shown by ray. Loud bronchial breathing after removal.
Fbdy. 1374—C-91 	3 yrs.	Peanut kernel	Left main bronchus, 6 weeks	None	5 mm. standard	Tight impaction with edema of mucous membrane affording forceps spaces only on deep inspiration. Friable fbdy.	Side-curved	Equator	Extraction Cure	9 min. 40 sec.	Oral	Some subglottic swelling. Mucosa of trachea and both bronchial orifices moderately inflammatory. Thick yellowish pus seen coming from left main bronchus and was aspirated. About one cm. below carina in left main bronchus, a ring of granulations and swollen mucosa encountered. A small amount of pus aspirated and peanut was seen between granulations.

Fbdy. 1376 	11 mos.	Peanut kernel	Left bronchus, at level of upper lobe, 6 days	None	4 mm. x 30 cm., standard	Swollen tissues; proximal annular edema; infant	Side-curved	Minor axis	Extraction Cure	7 min. 35 sec.	Oral	Child referred by pediatrist, Dr. Blechschmidt, who was consulted because of vomiting and intestinal disturbance. During a routine examination he discovered signs of obstruction in the left lung. Questioning of parents brought out history of child's choking on a piece of peanut candy. Secretions aspirated during bronchoscopy.
Fbdy. 1396—T-78  	18 mos.	Peanut kernel	Right main bronchus, 5 days	None	4 mm., standard	Presentation in pus and swollen mucosa; friable fbdy.; infant	Forward grasping	Minor axis	Extraction Cure	8 min. 15 sec.	Oral	Peanut kernel found lodged in right bronchus at level of upper lobe bronchus. Large amount of thin whitish pus aspirated prior to removal of peanut. Tracheal mucosa very inflammatory and a moderate amount of supraglottic swelling noted. Pus aspirated during bronchoscopy.
Fbdy. 1395—T-77  	2 yrs.	Peanut kernel	Right main bronchus, at level of upper lobe, 1 day	None	4 mm. x 30 cm., standard	Presentation. Friable fbdy.	Side-curved	Minor axis	Extraction Cure	3 min. 12 sec.	Oral	*First Bronchoscopy*: Thin, whitish pus coming up in large quantities through larynx on insertion of laryngoscope. Moderate subglottic swelling. Trachea very inflammatory. Large quantity of pus aspirated from trachea. Peanut found in right main bronchus at level of upper lobe. *Second Bronchoscopy* (one week later): Considerable inflammatory reaction in larynx and thin white pus coughed up freely from trachea which was quite inflammatory. Large piece of peanut kernel found in right main bronchus. Large quantity of pus aspirated from tracheobronchial tree prior to removal of peanut.

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1166	30 yrs.	Chicken bone	Esophagus, 4 days	None	Laryngoscope	Presentation of bone buried in pathologic tissue	Downjaw	One end	Extraction Cure	2 min. 35 sec.	Oral	Bone buried up to one end in anterior esophageal wall, surrounded by mass of sloughing and edematous mucosa. Endwise method of removal.
Fbdy. 1187—C-23	44 yrs.	Chicken bone	Esophagus below crossing of left bronchus, 5 days	None	9 mm. x 45 cm., standard	Presentation, one end buried	Rotation	One end, allowing the imbedded end to trail	Extraction Cure	7 min. 30 sec.	Oral	Bone transfixed across esophagus, right extremity imbedded. Rotation method of removal.
Fbdy. 1226	70 yrs.	Chicken bone	Esophagus just below cricopharyngeal fold, 9 days	None	9 mm. x 45 cm., standard	Finding	Plain	End	Extraction Cure	2 min. 30 sec.	Oral	Slender shaft rib bone found buried in esophageal wall. Area of infective esophagitis surrounded site of lodgment of bone. Endwise method of removal.

Fbdy. 1228	52 yrs.	Lamb bone	Esophagus below cricopharyngeus, 2 days	None	9 mm. x 45 cm., standard	Very large bone; tightly impacted	Rotation after turning with plain	Minor axis	Extraction Cure	30 min.	Oral	Version, followed by rotation method of removal.
Fbdy. 1280 	60 yrs.	Fish bone	Esophagus at level of crico pharyngeus, 5 days	None	9 mm. x 45 cm., standard	Finding in sloughing tissues	Alligator	End	Extraction Cure	6 min.	Oral	Large area of esophagitis just above cricopharyngeus and extending to a point above 3 cm. below cricopharyngeus. Small area sloughing; in this area a small spicule of sharp pointed hard substance, apparently fish bone, found buried. Endwise method of removal.
Fbdy. 1289—C42	37 yrs.	Bone	Esophagus at level crossing left bronchus, 24 hours	None	9 mm. x 45 cm., standard	Thick quadrangular bone	Rotation	One extremity	Extraction Cure	4 min. 15 sec.	Oral	Bone found transfixed across esophageal lumen at level of crossing left bronchus. The right extremity which was apparent was grasped with rotation forceps, the other extremity trailing. Rotation method of removal.

BONES—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1290—T-44	27 yrs.	Beef bone	Crosswise in pharyngoesophagus, 3 days	None	Laryngoscope	Crosswise fixation	Alligator	Upper free end	Extraction Cure	1 min. 15 sec.	Oral	Endwise method of removal.
Fbdy. 1296—T-45	43 yrs.	Pork bone	Esophagus upper thoracic constriction	None	9 mm. x 45 cm., standard	Crosswise fixation	Forward grasping after disimpaction of point by rotation	Free end	Extraction Cure	4 min. 20 sec.	Oral	Bone transfixed crosswise in esophagus just below cricopharyngeal fold. Sharp point deeply imbedded in right esophageal wall. Endwise method of removal, after version.
Fbdy. 1308—C-46	40 yrs.	Chicken bone	Esophagus suprasternal notch, 1 week	None	8 mm. x 45 cm., standard	Crosswise fixation	Rotation	One angle	Extraction Cure	6 min. 38 sec.	Oral	Bone found lying crosswise in upper part of thoracic esophagus in close apposition to anterior wall. Rotation method of removal.

Fbdy. 1343—T-55	45 yrs.	Chicken bone	Esophagus, at superior thoracic aperture, 2 days	None	9 mm. x 45 cm., standard	Crosswise fixation	Forward grasping	Flatwise at angle	Extraction Cure	4 min. 45 sec.	Oral	Points freed by lateral movement.
Fbdy. 1347—C-80	2½ yrs.	Chicken bone	Esophagus, below cricopharyngeus, 8 days	None	6 mm., standard	Presentation obscured by food	Rotation	Flatwise at irregular end	Extraction Cure	2 min. 30 sec.	Oral	Chicken bone in esophagus lying on posterior wall, partially obscured by food. Two points directed laterally with main portions projecting upward. Rotation method of removal.
Fbdy. 1353—T-62	65 yrs.	Fish bone	Esophagus below cricopharyngeus, 5 days	None	Adult esophageal speculum	Finding the small end of the bone that was outside esophageal wall in the pus and inflammatory folds. Withdrawal in the axis of penetration. Esophageal abscess and sloughing esophagitis.	Alligator	Free end	Extraction Cure	2 min. 46 sec.	Oral	Entrance of esophagus at level of cricopharyngeal fold was filled with foul pus. This was removed by aspiration and there was found imbedded in the left lateral esophageal wall below the cricopharyngeus, a large fish bone. This was removed, endwise. Mucosa of left upper esophageal wall inflammatory and swollen. At site of perforation of fish bone, a collection of pus had formed. This was evacuated by aspiration. Pus collection in retropharyngeal tissues required incision for aspiration, 13 days later.

BONES—*Continued*

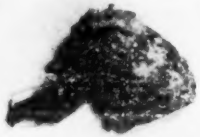
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1357—C-84	30 yrs.	Chicken bone	Esophagus, above hiatus esophageus, 3 days	None	9 mm. x 45 cm., standard	None	Rotation	Flatwise	Extraction Cure	7 min. 15 sec.	Oral	Several small erosions of esophageal mucosa 21 cm. from upper teeth. Chicken bone found above hiatus. It had evidently lodged for a time at the point indicated by the esophageal erosions, which corresponded closely to the point of pain complained of by patient. Rotation method of disimpaction and removal.
Fbdy. 1365—T-67	38 yrs.	Chicken bone	Esophagus at crossing of left bronchus, 1 day	None	9 mm. x 45 cm., standard	Crosswise fixation; obscuring of view by barium	Forward grasping and rotation	End nearest right esophageal wall	Extraction Cure	3 min. 12 sec.	Oral	Large mass of barium found lodged above chicken bone, removed with forward grasping forceps. Bone transfixed crosswise. Grasped with rotation forceps which brought away a fragment. Bone removed with forward grasping forceps. No evidence of trauma except at place where bone had perforated the mucosa.
Fbdy. 1382—C-96	50 yrs.	Chicken bone	Esophagus below crossing of left bronchus, 10 days	None	8 mm. x 45 cm., standard	Transfixed with one extremity perforating wall	Rotation	Near one end	Extraction Cure	6 min.	Oral	Bone transfixed in esophagus slightly below crossing of left bronchus. One extremity had perforated esophageal wall and many granulations were seen about this point.

Fbdy. 1384	28 yrs.	Chicken bone	Pharynx, 1 day	None	Laryngoscope	Prevention of escape downward	Alligator	First part seen	Extraction Cure		Oral	We deem it advisable to seize pharyngeal foreign bodies by any part first seen, regardless of mechanical problem, which is solved after seizure.
Fbdy. 1387—T-74 1387-T74	44 yrs.	Fish bone	Upper thoracic esophagus, 4 days	None	Adult esophageal speculum	Finding and presentation of small spicule of bone	Alligator	Free end	Extraction Cure	2 min. 31 sec.	Oral	Posterior pharyngeal wall and arytenoids edematous; cricopharyngeal fold red and swollen. Foreign body found projecting from right lateral wall of thoracic esophagus surrounded by inflammatory folds and purulent secretion.
Fbdy. 1392—C-101 1392-C101	37 yrs.	Fish bone	Esophagus below cricopharyngeus, about 7 hours	None	10 mm. x 45 cm., full lumen	Irregular serrated edge; edematous esophageal folds	Rotation	Flatwise at serrated end	Extraction Cure	11 min.	Oral	Marked inflammatory reaction found in esophagus at site of lodgment and surrounding area.

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1180	3 yrs.	Green pea	Esophagus, 1 day	None	7 mm. x 45 cm., standard	Foreign body in strictured lumen	Fenestrated	Bipolar	Extraction Cure	1 min. 30 sec.	Oral	Patient under treatment for cicatricial stenosis of the esophagus from lye. Whole pea found in lower stricture surrounded by a band of mild inflammation.
Fbdy. 1185	6 yrs.	Pea	Esophagus, 3 days	None	7 mm. x 40 cm., standard	Foreign body in cicatricially contracted lumen		Bipolar	Extraction Cure	3 min. 35 sec.	Oral	Patient had a cicatricial stenosis of esophagus. Pea lodged in former stricture caused by lye.
Fbdy. 1195	47 yrs.	Meat	Esophagus, below cricopharyngeus, 2 days	None	Forbes speculum	Foreign body not cohesive	T-shaped papilloma	As presented	Extraction Cure	4 min. 10 sec.	Oral	
Fbdy. 1200—C-28	24 yrs.	Food (two beans)	Above old stricture of esophagus, 2 days	None	9 mm. x 45 cm., standard	None	Plain	As presented	Extraction Cure	1 min. 43 sec.	Oral	Patient had lye stricture of esophagus. Food lodged while eating, causing temporary total obstruction and regurgitation. Beans crushed.

Fbdy. 1222	1 yr.	Egg shell	Esophagus, below cricopharyngeus, 3 days	None	Child's size laryngoscope	Friable foreign body	Alligator	Flatwise	Extraction Cure	5 min. 50 sec.	Oral	
Fbdy. 1238—C-31	20 yrs.	Meat	Esophagus (strictured)	None	8 mm., standard	No cohesion	Forward grasping	As presented	Extraction Cure	4 min. 20 sec.	Oral	Large mass of meat found immediately above strictured area.
Fbdy. 1256	20 yrs.	Meat	Esophagus (strictured), 1 day	None	9 mm. x 45 cm., standard	No cohesion	Meat	As presented	Extraction Cure	5 min. 30 sec.	Oral	Large bolus of imperfectly masticated food found tightly impacted in narrowing of lumen of middle third of esophagus. Chronic esophagitis present. This mass of meat would probably have lodged in a normal esophagus.
Fbdy. 1286—T-43	13 yrs.	Fruit pulp	Esophagus, 6 hours	None	6 mm. x 35 cm., standard	Friable mass of food	Forward grasping	As presented	Extraction Cure		Oral	Fruit pulp removed from esophagus during esophagoscopy for diagnosis. Esophagus found widely dilated.

FOODS—*Continued*

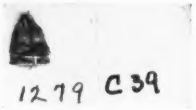
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1344—T-56 1344-T56	48 yrs.	Bolus of food	Esophagus, 25 cm. from upper teeth	None	7 mm. x 45 cm., standard	None	Forward grasping	As presented	Extraction Cure	6 min. 30 sec.	Oral	Bolus of food removed from organic narrowing of the esophagus during esophagoscopy for diagnosis
Fbdy. 1399—T-80  1399 - T80	50 yrs.	Bolus of meat	Esophagus, middle third, 1 day	None	7 mm. x 45 cm., standard	None	Side-curved	As presented	Extraction Cure	1 min.	Oral	Bolus of food had appearance of a smooth, hard foreign body. It was lodged in an organic cicatricial stenosis of middle third of esophagus.

BUTTONS

Fbdy. 1192—C-26 C26/192	12 yrs.	Campaign button	Esophagus, below cricopharyngeus, few hours	None	7 mm. x 45 cm., standard	Point of pin imbedded	Rotation	Flatwise at edge to produce version	Extraction Cure	8 min. 25 sec.	Oral	Point of pin imbedded in posterolateral wall. Rotation method of removal.

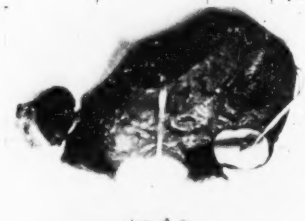
Fbdy. 1236	13 mos.	Patent button	Esophagus, below cricopharyngeus, 3 days	None	5 mm. x 30 cm., standard	Size of foreign body causing dyspnea in 13 month baby	Side curved	Small end	Extraction Cure	11 min. 30 sec.	Oral	Smaller end of button which seemed sharp, perforating the posterior wall of trachea and anterior wall of esophagus; pointed end of button sheltered in lip of tube-mouth for withdrawal.
Fbdy. 1244—C-104	18 mos.	Pearl button	Esophagus, suprasternal notch, about 2 weeks	None	5 mm. x 30 cm., standard				Foreign body passed into stomach, later passed by bowel. Cure	2 min. 12 sec.	By bowel	At esophagoscopy large bleeding irregular ulcer found in posterior pharyngeal wall on a level with right arytenoid. This bled very readily. Removal of foreign body postponed, awaiting healing of ulceration.
Fbdy. 1254	4 yrs.	Button	Esophagus, middle third, 6 months	None	5 mm. x 30 cm., standard	Grasping	Button	Flatwise	Extraction Cure	9 min.	Oral	Granulations found occluding esophagus in middle third; esophageal lumen very much narrowed. Patient became extremely dyspneic during removal. Dyspnea involved no risk because no anesthetic was used.
Fbdy. 1369—T 70	15 mos.	Button	Esophagus, suprasternal notch, 3 days	None	6 mm. x 30 cm., standard	Grasping button securely	Rotation	Flatwise, points in hole	Foreign body recovered in stools		Passed by bowel	Foreign body found at suprasternal notch, upper edge just below level of cricopharyngeal fold. Grasped with rotation forceps which failed to hold. On reinsertion of esophagoscope, foreign body passed on downward ahead of tube into stomach owing to ballooning of esophagus with air. Foreign body recovered in stools. Groove in button between the holes caused the point of the rotation forceps to slip off. Button forceps would have afforded secure hold.

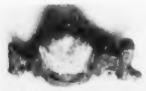
AMMUNITION

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1279—C 39 	4 yrs.	Bullet	Right stem bronchus, 1 month	None	5 mm., standard	Difficulty in getting forceps blades over minor axis, which was almost completely obscured by granulations, large quantity of pus	Forward grasping	Base of conoid	Extraction Cure	27 min. 15 sec.	Oral	Bullet almost completely obscured by granulation. When forceps first came in contact with foreign body a large quantity of pus gushed from around it. About two ounces of pus were aspirated.
Fbdy. 1304	3 yrs.	Cartridge blank	Left bronchus, below level of upper lobe, 8 days	None	5 mm., standard	Finding below acute inflammatory stenosis	Forward grasping, heavy	Rim edge	Extraction Cure	6 min. 35 sec.	Oral	Left bronchus swollen shut on expiration. On inspiration the bronchial lumen opened up slightly and air passed both in and out but the lumen was insufficient to see through. Forceps inserted and placed under the direction of the roentgenologist, Dr. Magoes. Proximal (rim) edge of cartridge shell seized and withdrawn along with bronchoscope.
Fbdy. 1355—T-63	5 yrs.	Bullet	Right main bronchus, level upper lobe, 5 days	None	6 mm., standard	Absence of forceps spaces. Secure grasping	Forward grasping, with deeply cupped blades, heavy	Opposite poles	Extraction Cure	13 min. 33 sec.	Oral	Slight inflammatory reaction in larynx and trachea with mucopus coming up from right bronchus. Pus was removed by aspiration. Considerable inflammatory swelling with slight edema of right main bronchus. No abrasions.

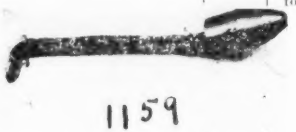
Fbdy. 1174 1174	19 yrs.	Artificial denture with 3 teeth attached	Esophagus, below cricopharyngeus, 4 days	None	9 mm. x 45 cm., standard	Large impacted foreign body. Avoidance of trauma	Rotation		Extraction Cure	4 min. 30 sec.	Oral	Foreign body found in ulcerated, lacerated area below swollen folds about 5 cm. below cricopharyngeus.
Fbdy. 1264	21 yrs.	Artificial denture	Esophagus, lower end, 8 days	None	9 mm. x 45 cm., standard esophagoscope and Forbes speculum	Large, sharp impacted foreign body. Avoidance of trauma	Rotation	Various points in succession as needed for version	Extraction Cure	14 min. 33 sec.	Oral	Tooth plate found at lower end of esophagus giving appearance of being unable to go through hiatus. Intense esophagitis and ulceration of tissue in neighborhood of sharp edges of tooth plate. Seized with rotation forceps and rotated so as to come up sidewise until under surface of cricopharyngeal fold was reached. Forbes speculum then substituted for esophagoscope; through this, folds disengaged from foreign body after seizure with rotation forceps, and foreign body removed.
Fbdy. 1274 1274	26 yrs.	Tooth	Right lower lobe bronchus, 3 years	Local	5 mm. x 40 cm., standard	Finding tooth at level of diaphragm (40 cm. from upper teeth) in a laterally deviating fistula. Dilatation of stricture. All movements of adherent diaphragm communicated forcibly to lung	Plain, heavy, strongly dilating	One end	Extraction Cure	57 min. 27 min. 25 sec. 8 min. 15 sec. 25 min. 28 min. 10 sec.		Unsuccessful attempt to remove tooth by external operation one month before admission. Four bronchoscopies required for dilatation of fistula and drainage of lung before foreign body could be reached. Removed at fifth bronchoscopy.

DENTAL AND SURGICAL OBJECTS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1303 	30 yrs.	Tooth plate	Esophagus, upper end, level of suprasternal notch, 12 days	None	10 mm x 45 cm., standard	Very tight impaction in edematous tissue. Bed of tooth plate ulcerated	Rotation	Near edge	Extraction Cure	29 min. 20 sec.	Oral	Esophagus at the level of arch of aorta found to be extremely edematous and almost closed with swelling. Repression of these swollen folds revealed the upper edge of the tooth plate which was too wide for removal because of the projection of one tooth. This one tooth was detached and removed with a small portion of the plate. Balance of plate then turned and removed.
Fbdy. 1346—T-58	22 yrs.	Drainage tube	Left pleural cavity, 1 day	None	Pleuroscope	Presentation	Forward grasping	Edge of end of tube	Extraction Cure of foreign body condition.	1 min. 38 sec.	Thoracotomy fistula (old)	Tube found just within the outer wall of cavity on diaphragmatic surface.

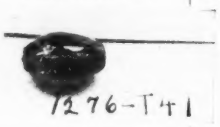
Fbdy. 1377—C-92  1377-C92	34 yrs.	Gold tooth bridge	Left main bronchus. 12 days	Local	9 mm. x 40 cm.	Difficulty in dislodging distal extremity from left upper lobe bronchial orifice. With forceps and lip of bronchoscope axis traction was made possible	Rotation	At upper end near inner edge	Extraction Cure	4 min. 10 sec.	Oral	At bronchoscopy gold bridge seen in left main bronchus with distal extremity extending into orifice of left upper lobe bronchus. Several small granulations present on inner wall at level of proximal end of bridge.
Fbdy. 1391—T-76 1391-T76	48 yrs.	Artificial denture	Hypopharynx, 5 hours	None	Adult laryngoscope	Presentation of end and rotation	Rotation	Right posterior corner	Extraction Cure		Oral	Broad end of tooth plate downward, lower surface anterior. Single tooth which was attached to plate was first portion encountered.
Fbdy. 1394 1394	49 yrs.	Tooth filling	Upper portion, right lower lobe, 4 weeks	None	8 mm. x 40 cm., standard	Finding, in bleeding granulation tissue	Forward grasping	Minor axis	Extraction Cure	3 min. 45 sec.	Oral	Right stem bronchus found completely occluded at level of the middle lobe bronchial orifice by granulation tissue and swollen mucosa, both of which practically occluded the middle lobe bronchus. After aspirating a large quantity of pus, foreign body found buried in granulation tissue in upper portion of lower lobe bronchus.

DENTAL AND SURGICAL OBJECTS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1397	27 yrs.	Tooth filling	Right stem bronchus, just below orifice of middle lobe bronchus, 3 months	None	8 mm. x 40 cm., standard	Finding, in pathologic tissue	Forward grasping	Minor axis	Extraction Cure	26 min. 35 sec.	Oral	Foreign body found imbedded in granulation tissue.
TOYS												
Fbdy. 1159 	6 yrs.	Metallic object from toy	Right main bronchus, 8 days	None	6 mm. x 35 cm., aspirating	Shape of object, anchorage in swollen mucosa	Side-curved	End	Extraction Cure	5 min. 20 sec.	Oral	Upper end of foreign body found at bifurcation; distal portion imbedded and anchored below orifice of middle lobe bronchus in stem of right lower lobe bronchus.
Fbdy. 1175—C-18	7 yrs.	Ear from toy horse	Left main bronchus, 2 days	None	6 mm. x 35 cm., standard		Side-curved	Minor axis	Extraction Cure	3 min. 10 sec.	Oral	Small amount of secretion present.

Fbdy. 1194 1194	5 yrs.	Whistle	Esophagus, at arch of aorta, 1 day	None	7 mm., standard	Presentation	Rotation	Flatwise, forceps points in holes	Extraction Cure	3 min. 38 sec.	Oral	Rotation forceps held well in central holes of fbdy
Fbdy. 1223 1223.	10 mos.	Toy goat	Esophagus, below cricopharyngeus, 10 hours	None	Child's size laryngoscope		Alligator		Extraction Cure	3 min. 10 sec.	Oral	Legs of toy upward.
Fbdy. 1224 1224	13 mos.	Tin steering wheel from toy automobile	Esophagus, about 3 months	None	Child's size laryngoscope used as hypopharyngoscope	Old pathology	Alligator	Flatwise	Extraction Cure	18 min.	Oral	Foreign body found below swollen masses of edematous tissue, buried in granulations and foul sloughs. Rough edges of tin had caused ulceration.

TOYS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1276—T-41 	4 yrs.	Bead	Left lower lobe bronchus, 1 day	None	5 mm. x 30 cm., standard	Securing a firm hold on bead	Stiff spring, forward grasping, cupped, heavy	Minor axis	Extraction Cure	14 min. 29 sec.	Oral	On insertion of bronchoscope, bead was coughed against tube-mouth and immediately reaspirated. Inspection of the right bronchial orifice showed abrasion and inflammatory reaction of the mucosa corresponding to localization by physical signs and X-ray of the location of foreign body. Bead was removed from left lower lobe bronchus.
Fbdy. 1352—C 82	13 mos.	Jackstone, toy (cast iron)	Esophagus, upper third, 3 weeks	None	Child's size laryngoscope	Object imbedded in edematous, sloughing folds	Cupped	Presenting knob grasped; folds disengaged by gentle rocking and rotation	Extraction Death	2 min. 35 sec.	Oral	Child treated for bronchitis for 3 weeks before admission, notwithstanding foreign body history. Was also thought to have diphtheria. Jackstone discovered in esophagus by X-ray after admission. Child very ill on admission. Intense dyspnea, with marked inspiratory indrawing. Child seemed exhausted. Cervical cellulitis. Tracheotomy performed for dyspnea immediately after admission. Esophagoscopy, 3 days after tracheotomy. The larynx was swollen completely shut. Jackstone seen in the esophagus below cricopharyngeus with two knobs presenting, left one the nearer. Marked edematous esophagitis present. Knob grasped with cupped forceps and by gentle rocking and rotating, foreign body was disengaged from the folds and removed. Child seemed quite well for about seven days after esophagoscopy. Then refused to take its feedings. On the 11th day after removal of foreign body patient had two severe hemorrhages and died. No autopsy was obtained. It is believed that the bleeding came from the point of lodgment of the foreign body which undoubtedly was ulcerated.

Fbdy. 1362—C 87 1362-C87	4 yrs.	Bead	Right main bronchus, 2 days	None	5 mm. x 30 cm., standard	Difficulty in getting forceps over minor axis for secure grasping	Cupped, forward grasping	Minor axis	Extraction Cure	10 min. 54 sec.	Oral	Marked subglottic swelling with intense inflammatory reaction of mucosa of trachea and of bronchial orifices. Bead was seen in right main bronchus, large extremity uppermost. It was necessary to grasp the bead several times before a secure grasp was obtained. On removal foreign body was stripped off in pharynx and swallowed by patient. Recovered in stools.
Fbdy. 1388—T 75 1388-T75	9 yrs.	Reed from toy saxophone	Right stem and lower lobe bronchus	None	6 mm. x 35 cm., standard	None	Forward grasping	Minor axis	Extraction Cure	2 min. 8 sec.	Oral	Pointed end of foreign body upward in right stem bronchus, the upper end being in right stem bronchus at level of middle lobe bronchus.
Fbdy. 1389—C 100 1389-C100	5 yrs.	Tin whistle	Esophagus, below cricopharyngeus, 7 days	None	Child's size Forbes speculum	Inflammatory swelling with granulations	Rotation	Flatwise, forceps points in holes of fbdy.	Extraction Cure	1 min. 30 sec.	Oral	Marked localized esophagitis; granulation tissue on anterior esophageal wall.

RUBBER AND WOOD

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1242 1242	5 yrs.	Portion of rubber auto tire	Right lower lobe bronchus, 6 weeks	None	5 mm. x 30 cm., standard	Finding	Mosquito	Flatwise	Extraction Cure	3 min. 59 sec. 14 min. 40 sec.	Oral	*First Bronchoscopy*: Right bronchus very inflammatory with small granulomatous mass projecting from anterior wall; mucosa granular; some mucopus present; no foreign body seen. Left bronchus obstructed apparently by collapse during cough and suppressed breathing. *Tracheotomy* for dyspnea necessary 24 hours after bronchoscopy. Patient coughed up large quantity of very thick tenacious mucus, when trachea was opened. *Second Bronchoscopy* (per oral): Mass of granulation tissue and copious pus discharge found in right lower lobe. After aspiration and removal of granulations a small object was seen entirely filling a branch of the right lower lobe bronchus.
Fbdy. 1210—T 29 1210-T29	10 mos.	Fragment of wood	Trachea near bifurcation, 1 day	None	4 mm. x 30 cm., standard	Grasping; rotating so that wedge would pass through glottis	Side-curved	One end	Extraction Death	3 min. 25 sec.	Oral	Pharynx filled with mucopus coughed up from trachea. Lumen of larynx was abnormally small, admitting 4 mm. bronchoscope at tight fit. Trachea filled with pus. Foreign body removed. Child was much easier following bronchoscopic removal and was able to swallow liquids; but subsequently grew worse. Tracheotomy and aspiration gave some relief but child died of bronchopneumonia.

Fbdy. 1249—T-36 1249 T 36	9 mos.	Splinter of wood	Pharynx, about 40 hours	None	Child's size laryngoscope	Grasping and pushing downward to disengage point from pharyngeal wall	Alligator	Upper end of foreign body presenting	Extraction Death	3 min. 24 sec.	Oral	On admission child very ill; dyspneic; indrawing. Emergency tracheotomy performed, without waiting to determine cause of dyspnea or presence of foreign body. Larynx then exposed with direct laryngoscope. No foreign body seen; tip of laryngoscope inserted in right pyriform sinus and larynx pulled gently forward. Black object seen in pharynx back of larynx. On deep insertion of laryngoscope it was found that upper end of object was imbedded in posterior pharyngeal wall, the lower end projecting forward impinging trachea just below larynx. Point of foreign body disembedded, and object removed. Found to be a splinter of wood, triangular in shape. Child showed progressive improvement for several days following removal of foreign body. Developed bronchopneumonia. Death, 21 days after removal of foreign body.

COINS AND OTHER DISCS

Fbdy. 1158 1158	2½ yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 19 days	None	7 mm. x 30 cm., retrograde	Presentation	Side curved	Flatwise	Extraction Cure	1 min.	Oral	Coin corroded and covered with grayish colored food.

COINS AND OTHER DISCS—*Continued*

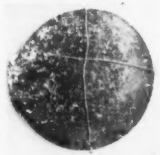
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1165—C-17	3 yrs.	Coin (penny)	Esophagus, below cricopharyngeus, 5 weeks	None	Anterior commissure laryngoscope	Presentation	Alligator	Flatwise	Extraction Cure	1 min. 15 sec.	Oral	Moderate degree of esophagitis present.
Fbdy. 1167—T-20	2 yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 9 days	None	7 mm. x 45 cm., standard	Presentation. Coin covered with food debris	Forward grasping	Flatwise	Extraction Cure	5 min. 7 sec.	Oral	Coin covered with secretions and particles of food.
Fbdy. 1171	3 yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 5 days	None	7 mm. full lumen	Presentation	Side-curved	Flatwise	Extraction Cure	39 sec.	Oral	
Fbdy. 1172—T-22	4 yrs.	Two metal discs	Esophagus, below and behind cricopharyngeal	None	7 mm. x 40 cm.,	Presentation	Forward grasping	Flatwise	Extraction Cure	9 min., 35 sec.	Oral	

Fbdy. 1172—T-22	4 yrs.	Two metal discs	Esophagus, below and behind cricopharyngeal fold, 1 day	None	7 mm. x 40 cm., standard	Presentation	Forward grasping	Flatwise	Extraction Cure	9 min., 55 sec.	Oral	
Fbdy. 1176—T-23	3 yrs.	Coin (nickel)	Esophagus, below cricopharyngeal fold, 11 days	None	Anterior commissure laryngoscope	Presentation	Downjaw	Flatwise	Extraction Cure	2 min. 35 sec.	Oral	
Fbdy. 1181—C-20	2 yrs.	Coin (penny)	Esophagus, below cricopharyngeus, 6 days	None	Small Forbes esophageal speculum	Presentation	Alligator	Flatwise	Extraction Cure	50 sec.	Oral	Coin obscured by fold of mucosa.

COINS AND OTHER DISCS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1186—C-22 1186-C22	7 yrs.	Coin (quarter)	Esophagus, below cricopharyngeus, 5 days	None	7 mm. x 45 cm., standard	Swollen folds; impaction	Side-curved	Flatwise	Extraction Cure	2 min. 59 sec.	Oral	Coin hidden behind folds of cricopharyngeus.
Fbdy. 1188—C-24 1188-C24	3 yrs.	Coin (quarter)	Esophagus, below cricopharyngeus, 5 days	None	7 mm. x 45 cm., standard	Coin was firmly anchored below cricopharyngeus	Side-curved	Flatwise	Extraction Cure	6 min. 35 sec.	Oral	Coin hidden behind folds of cricopharyngeus.
Fbdy. 1227 1227	6 yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, about 6 hours	None	7 mm. x 45 cm., standard	Presentation	Plain	Flatwise	Extraction Cure	1 min.	Oral	

Fbdy. 1232

Fbdy. 1232 	5 yrs.	Metal disc (cap)	Esophagus, level suprasternal notch, 1 year	None	Anterior commissure laryngoscope	Prevention of trauma	Down-jaw		Extraction Cure	4 min. 15 sec.	Oral	Metal cap found imbedded in posterior wall, the rim of cap having cut its way deeply into mucosa like a biscuit cutter; mucosa sloughing; odor very foul.
Fbdy. 1241—T-34	7 yrs.	Coin (quarter)	Esophagus, just above diaphragm, 5 hours	None	7 mm. x 40 cm., gastroscope	Edge of coin was presented with lip of tube between folds of stomach	Forward grasping	Flatwise	Extraction Cure	3 min. 20 sec.	Oral	Coin seen just above level of diaphragm. On approaching with esophagoscope it disappeared through hiatus and was followed into stomach from which it was promptly removed with esophageal forceps.
Fbdy. 1248—C 33	2 yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 1 week	None	Child's size Forbes speculum	None	Down jaw	Flatwise	Extraction Cure	1 min. 55 sec.	Oral	Upper margin of coin covered by cricopharyngeus.

COINS AND OTHER DISCS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1253—C-34	2 yrs.	Coin (nickel)	Esophagus, just above level suprasternal notch, 4 days	None	Forbes speculum	Presentation	Alligator	Flatwise	Extraction Cure	2 min. 45 sec.	Oral	Upper margin of coin hidden below cricopharyngeus.
Fbdy. 1257—T-37	1 yr	Coin (penny)	Esophagus, below cricopharyngeal fold, 6 days	None	Child's size laryngoscope	Removal of food debris and presentation	Alligator	Flatwise	Extraction Cure	1 min. 20 sec.	Oral	Coin covered with food debris.
Fbdy. 1259—C-35	18 mos.	Coin (penny)	Esophagus, below cricopharyngeus, 6 days	None	Child's size Forbes speculum	Presentation	Alligator	Flatwise	Extraction Cure	1 min. 20 sec.	Oral	Covered with food particles.

Fbdy. 1271—T-33	5 yrs.	Coin (penny)	Esophagus, below cricopharyngeus, 5 hours	None	Child's size Forbes speculum	None	Alligator	Flatwise	Extraction Cure	3 min. 27 sec.	Oral	
Fbdy. 1275—T-40	3 yrs.	Dress weight	Esophagus, level suprasternal notch, 15 days	None	6 mm. x 35 cm., standard	Presentation in granulation and inflamed folds	Forward grasping	Flatwise	Extraction Cure	13 min. 30 sec.	Oral	Cervical esophagus filled with foul smelling pus. Mucosa granular and fungating, due to sojourn of foreign body. Foreign body covered with granulations and food debris.
Fbdy. 1278—C-38	3 yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 9 days	None	Child's size Forbes speculum	Obscured by food and inflammatory folds	Alligator	Flatwise	Extraction Cure	3 min. 40 sec.	Oral	Considerable inflammatory reaction below cricopharyngeus.

COINS AND OTHER DISCS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1285—C-41	5 yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 1 day	None	Child's size Forbes speculum	Presentation	Alligator	Flatwise	Extraction Cure	1 min. 50 sec.	Oral	Upper edge of coin hidden in cricopharyngeal fold. This child had a penny removed from the esophagus about two years previously. Fbdy. No. 1029.
Fbdy. 1287	3 yrs.	Coin (penny)	Esophagus, below cricopharyngeus, 5 days	None	7 mm. full lumen	Presentation	Plain	Flatwise	Extraction Cure	48 sec.	Oral	
Fbdy. 1300—T-46	7 yrs.	Coin (quarter)	Esophagus, at crossing of left bronchus, 8 hours	None	6 mm. x 35 cm., standard	Presentation; avoidance displacement downward	Forward grasping	Flatwise	Extraction Cure	1 min. 52 sec.	Oral	Coin found lodged just below cricopharyngeal fold. On introduction of esophagoscope coin moved downward to level of crossing of left bronchus.

Fbdy. 1307—C-45 1307-C45	2½ yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 7 days	None	Child's size Forbes speculum	Presentation	Alligator	Flatwise	Extraction Cure	3 min. 41 sec.	Oral	Upper edge of coin hidden behind cricopharyngeal muscle.
Fbdy. 1313—C-48 1313-C48	6 yrs.	Coin (penny)	Esophagus, at crossing of aorta above esophageal stricture, 1 day	None	7 mm. x 45 cm., standard		Side grasping	Flatwise	Extraction Cure	2 min 17 sec.	Oral	Lye stricture of the esophagus.
Fbdy. 1315—C-49 1315-C49	3 yrs.	Coin (quarter)	Esophagus, behind manubrium, 5 months	None	7 mm. x 30 cm., full lumen	Presentation, swelling and bleeding granulations from 5 months' sojourn	Side-curved	Flatwise	Extraction Cure	2 min. 9 sec.	Oral	Roentgen ray examination a week after coin was swallowed was said to have failed to reveal the coin. Tonsillectomy had been advised to relieve difficulty in swallowing. Child developed symptoms of bronchitis with fever, cough, etc. X ray 4 months after accident revealed coin in esophagus. At esophagoscopy coin found free in esophagus slightly below granulating area. It seems probable that the coin had been lying at the point where the pathology was evident, and that it had recently left this location and lodged further down. Pathology evidently due to prolonged sojourn.

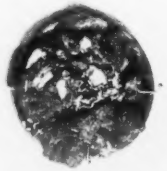
COINS AND OTHER DISCS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1316	11 yrs.	Coin (half dollar)	Esophagus, just below cricopharyngeus, 2 days	None	9 mm. x 45 cm., standard	Tight impaction below cricopharyngeus	Side grasping	Flatwise	Extraction Cure	8 min. 52 sec.	Oral	Moderate inflammatory reaction.
Fbdy. 1318—C 50	4 yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 3 days	None	Child's size Forbes speculum	Presentation	Down-jaw	Flatwise	Extraction Cure	1 min. 38 sec.	Oral	Upper border of coin hidden by cricopharyngeal fold.
Fbdy. 1338—C 76	18 mos.	Coin (nickel)	Esophagus, at cricopharyngeus, 4 weeks	None	7 mm. x 45 cm., standard	Presentation; inflammatory folds	Side-curved	Flatwise	Extraction Cure	2 min. 35 sec.	Oral	Coin hidden in folds of cricopharyngeus which were seat of inflammatory process.

Fbdy. 1340—C 77	2½ yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, few hours	None	7 mm. x 45 cm., standard	Presentation	Side-curved	Flatwise	Extraction Cure	2 min. 45 sec.	Oral	Upper margin of coin hidden by cricopharyngeal folds.
Fbdy. 1348—T 59	2½ yrs.	Coin (quarter)	Esophagus, below cricoid level, 5 days	None	Child's size Forbes speculum	Presentation	Down jaw alligator	Flatwise	Extraction Cure	3 min. 21 sec.	Oral	Coin covered with food debris and secretion, posterior to cricopharyngeal fold.
Fbdy. 1350—C 81	3 yrs.	Coin (quarter)	Esophagus, below cricopharyngeus, 6 days	None	7 mm. x 45 standard	Presentation	Forward grasping	Flatwise	Extraction Cure	2 min.	Oral	Upper edge of coin hidden by cricopharyngeal fold.

COINS AND OTHER DISCS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1356—T-64 1356-T64	3 yrs.	Coin (penny)	Esophagus, below cricopharyngeus, 5 hours	None	7 mm. x 45 cm., standard	Avoiding dislodging downward	Forward grasping	Flatwise	Extraction Cure	1 min. 7 sec.	Oral	On presentation of the lip of the esophagoscope the penny was displaced downward in esophagus to level of diaphragm from which point it was removed.
Fbdy. 1359—C-86 1359-C86	1 yr.	Coin (penny)	Esophagus, below cricopharyngeus, 7 days	None	Child's size Forbes speculum	None	Alligator	Flatwise	Extraction Cure	1 min. 30 sec.	Oral	Upper margin of coin hidden behind cricopharyngeus. Some erosions along right side of esophagus.
Fbdy. 1361—T-66 1361-T66	7 yrs.	Coin (quarter)	Esophagus, below cricopharyngeus, 30 hours	None	7 mm. x 45 cm., standard	None	Forward grasping	Flatwise	Extraction Cure	32 sec.	Oral	

Fbdy. 1372—C-89  1372-C89	2½ yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 5 days	None	Child's size Forbes specu-lum	Obscured by food	Alliga-tor	Flatwise	Extraction Cure	2 min. 10 sec.	Oral	Large quantity of food in esophagus at cricopharyn geus. After removal of this, coin and adherent food removed.
Fbdy. 1373—C-90 C90-1373	2 yrs.	Coin (penny)	Esophagus, below cricopharyngeus, 1 week	None	6 mm. x 35 cm., stand ard	Obscured by food. Swol-len esopha-geal folds	Side-curved	Flatwise	Extraction Cure	6 min. 30 sec.	Oral	Considerable quantity of food adherent to coin. A few granulations seen immediately above coin.
Fbdy. 1375—T-72 1375-T72	4 yrs.	Coin (nickel)	Esophagus, at suprasternal notch, 2 days	None	6 mm. x 35 cm., stand ard	Presentation and grasp-ing	Forward grasp ing	Flatwise	Extraction Cure	3 min. 15 sec.	Oral	On insertion of esophago scope, coin retreated to just above diaphragm from which point it was removed.

COINS AND OTHER DISCS—*Continued*

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1380—T-73	18 mos.	Coin (nickel)	Esophagus, below cricopharyngeal fold, 10 days	None	6 mm. x 35 cm., standard	Presentation	Forward grasping	Flatwise	Extraction Cure	8 min. 5 sec.	Oral	Folds of esophagus inflammatory and swollen.
Fbdy. 1386—C-99	2 yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 2 weeks	None	Child's size Forbes speculum	None	Alligator	Flatwise	Extraction Cure	55 sec.	Oral	Coin almost hidden by particles of food.
Fbdy. 1400—C-102	3 yrs.	Coin (nickel)	Esophagus, below cricopharyngeus, 2 months	None	Child's size Forbes speculum	Large amount of bleeding granulation tissue completely hiding coin	Alligator	Flatwise	Extraction Cure	6 min. 10 sec.	Oral	Coin completely covered with granulations.

III.

A MICROSCOPIC STUDY OF THE EXCISED TONSIL IN RELATION TO TONSILLECTOMY.

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The literature abounds in a vast amount of data regarding the many methods of tonsil removal, each method laying claim to certain points of superiority through the accomplishment of certain essential features, but there is an almost entire absence, in the laryngologic literature, of any definite comparative study, in so far as it pertains to the histologic aspect of the subject, in relation to the various methods. It is in view of the clinical interest involved that this investigation was undertaken.

From the microscopic study of the excised tonsils I wished to demonstrate: (1) The existence or nonexistence of the so-called true "tonsil capsule." (2) To show whether or not the removal of the tonsil could take place without injury to or removal of some adjacent voluntary muscle tissue. (3) The influence of the latter on the end result of the healing, scar tissue formation and deformity, and lastly (4) To briefly shed some light on the socalled bloodless tonsillectomy.

Reviewing the anatomy of the tonsils, we find that they are oval masses of lymphoid tissue, situated one on each side in the recess between the anterior and posterior palatine arches. Their long axes are directed from above downwards and somewhat backwards, reaching from the soft palate above to the side of the tongue below. They measure at puberty on an average of 20 to 25 mm. vertically, 10 to 15 mm. from before backwards, and from 5 mm. to 10 mm. within outward. The anterior boundary of this recess is formed by the palatoglossus muscle, the palatopharyngeus forming the posterior boundary, and the outer, or attached surface, contains fibers of the superior constrictor and internal pterygoid muscles, and the lower, the fibrous tissue of the tongue. Separating the tonsil from these surrounding muscle fibers are aponeu-

rotic fascia and connective tissue, and the area of this attached surface of the gland is dependent upon the development present in the individual case.

For this study 172 tonsils were utilized, or seven times twelve pairs, removed by as many different methods by laryngologists of recognized skill in various parts of America. Later two additional pairs of tonsils were added, representing still another method. These tonsils fell into the two general classifications, viz., large, or hypertrophied, and small, or atrophic, type.

The tonsils immediately after removal were placed in a 4 per cent formalin solution and sent to us. They were then imbedded in paraffin and macroscopic, but, more particularly, histologic and microscopic, study of the material was made. For the histologic work approximately 500 sections were prepared and an average of three sections from each tonsil cut and examined. From every specimen at least one section was made to pass through the long axis—that is, from the superior to the inferior pole. Further sections were made, so as to include parts of the surfaces that gave the gross appearance of having muscle attached to them. In explanation of this manner of selecting material from which to prepare sections, we refer the reader back to what was given as the purpose of this study.

Because of the various types of operation and instruments employed in removing the specimens under investigation, it was found advisable to divide the eight sets into as many groups as the method or type employed in each case. I have, therefore, tabulated the findings, giving first place to the phase of the work chiefly concerning us, but in addition noting pathologic changes in the material examined.

From the study of the many specimens from which the sections were made, I am convinced that the term "capsule," as it would apply to the ordinary gland or kidney, is, from a descriptive standpoint, most misleading and serves no other purpose, as Patterson has said, than to be merely a surgically convenient term, for in no instance was it possible to demonstrate anything similar.

It was found that the aponeurosis and connective tissue separating the tonsil from its muscle bed is quite variable in

amount and distribution, in some instances appearing as only a very thin "stripe" and lacking uniformity in thickness throughout its attached surface. Part of this connective tissue continues on into the tonsillar structure for some distance as septa, or divisions, of the gland.

Presence of Muscle Tissue in Removed Tonsils.—Only a few, twelve in all, of the series showed microscopically no muscle tissue attached (in one of these the complete removal being doubtful), regardless of which method of tonsillectomy was employed. Of 124 tonsils removed by the guarded, or ring type of instrument, whether containing a blade or snare wire, 77 showed various amounts of muscle tissue present, 29 traces of muscle tissue, and only 6 no muscle tissue. Of the latter group, complete removal was doubtful in one specimen.

In the dissection method, of 48 tonsils examined that had been removed by two different operators, 30 showed muscle tissue in various quantities, 13 traces of muscle tissue, and 3 no muscle tissue where complete removal had taken place.

It was found that, as a general rule, the smaller the tonsil, especially when the seat of a chronic interstitial or a chronic peritonsillitis, the greater was the amount of muscle brought away with it. However, in the case of larger specimens with little or no muscle attached to the lateral surface, frequently did we see a collection of muscle bundles at the anterior or posterior margin, or a point corresponding to the position of the glossopalatine or pharyngopalatine arch. In the majority of sections the source of the muscle tissue present was from the palatoglossus muscle of the anterior pillar.

Explanation of the frequency with which muscle tissue is found suggests the following: (1) The nonexistence of a so-called true tonsil capsule, thereby rendering a separation of the connective tissue fibers from the aponeurosis the rarest of accomplishments, the success of which is influenced to a large degree by the anatomic development in the given case. (2) The size and character of the tonsil. In children with large or hypertrophied tonsils, smaller amounts of muscle tissue were present than in adults with small and chronically inflamed tonsils, due, no doubt, to the usual absence of long standing peritonsillar inflammation in the former, thereby

rendering the tonsil less adherent. (3) The protection offered the adjacent mucous membrane and muscle structures by the instruments of the ring, or square-opening type, wherein is lodged the snare or blade, is not complete, regardless of the skill or the experience of the operator. The fact that with instruments of this type the tonsil is required to be pushed through the opening before the snare wire or blade is brought home is sufficient frequently to bring about the presentation of some muscle tissue and result in its removal.

The part of this report indicating whether or not there had been a complete removal of the tonsil was based on a study of carefully prepared sections taken from various parts of the attached surface and the anterior and posterior margins. Mindful of the fact that there may be some disagreement as to the extent to which this method of determination of complete removal can be relied upon, it would seem that properly prepared sections, subjected to careful study, should yield dependable results.

It is interesting to note in this connection that as a general thing the surgical method credited with the greater number of complete removals is charged with a higher percentage of muscle attached. On the other hand, the smaller the number with muscle attached, the greater the percentage of cases in which a part of the tonsil was not removed with the larger part of the tonsils sent us for study.

As will be seen from the table, the number of tonsils removed by the dissection method which showed muscle tissue in the sections is as high or slightly higher than the average of specimens removed by operations wherein the ring type of instrument was used. These findings might be misleading without the explanation that in all but a very few of these cases the muscle tissue present was in the form of mere tags, hardly more than enough to be recognized as such. Another interesting point in connection with sections from this group is seen in the fact that little or no muscle was found occupying a place corresponding to the position of the anterior or posterior pillars of the fauces, and the mucous membrane with the epithelium covering these structures was represented in but few of the sections. Also the dissection method gave us the greatest number of complete removals.

The appended tabulative outline of our findings would indicate that but few tonsils are removed without some adjacent muscle tissue attached.

The importance of conservation of the adjacent muscle tissue of the tonsils, together with its mucous membrane covering, as it passes from the surface of the tonsil to the glossopalatine and pharyngopalatine arches is apparent, for when the palatoglossus and pharyngeus forming the anterior and posterior pillars are subjected to the minimum amount of injury and these edges remain covered with mucous membrane from which epithelization can take place more readily, convalescence is accomplished over a shorter period of time. I am of the opinion that the occurrence of deformity in the operative area is greatly influenced by the amount of muscle tissue removed at the time of operation, with an added factor when the margins of the anterior and posterior pillars are stripped bare of all mucous membrane.

In such cases healing or convalescence must be prolonged. Complications are more apt to arise and the wound heals by granulation and scar, resulting frequently in variable degrees of deformity, and is accountable, no doubt, for much of the discomfort from which many of these patients complain. The granulation tissue may persist over a long period and be the site of repeated attacks of inflammation, which, when so inflamed, gives the appearance of tonsillar remnants from a previous operation.

Bleeding.—In this study I have also been mindful of the question of bleeding and the influence upon it by the various methods of operation and instruments. I am convinced that, owing to the anatomic differences existing in each case, no instrument or method has yet been devised that can offer any assurance of freedom from bleeding. In some of the specimens have been found blood vessels of considerable size entering the tonsils, and in others, vessels quite small, entering the tonsils at various angles and locations. The amount of bleeding produced by any given method of removal or instrument is chiefly influenced by the anatomic development in the given case.

That the question of muscle fibers attached to the excised tonsil is of no ultimate clinical importance, I am convinced.

when they are only fragmentary; but feel that something can be usefully added to the subject of tonsillectomy by this investigation, if only it serves the purpose of inviting more careful attention to one of the highly important points in accomplishing the satisfactory end results of tonsillectomy that has heretofore not received the consideration its importance should demand.

CONCLUSIONS.

1. Comparatively few tonsils are removed without some muscle tissue attached.

2. The largest quantity of muscle tissue appeared in sections of the smaller, or atrophic type of tonsils in adults. In these cases the muscle was found attached mainly to the lateral surface.

3. In the case of the larger tonsils, especially in children, the muscle tissue consisted chiefly of part of the palatoglossus and the mucous membrane of the anterior pillar.

4. A socalled true capsule of the tonsil, which can be separated as a definite independent structure, does not exist.

5. The amount of bleeding and type of bleeding are dependent chiefly on the anatomic development and distribution of the blood supply in the individual case and not upon the instrument used for tonsillectomy.

6. The amount of postoperative discomfort and deformity in end results are influenced by the amount of adjacent muscle tissue removed at the time of operation.

7. Small fragmentary fibers of muscle tissue are impossible to avoid at the time of operation and are of no clinical consequence or importance.

8. The dissection operation properly performed and under direct view, step by step, as the tonsil is enucleated, yields the greater number of complete removals.

I am under obligation to Dr. Henry C. Hartman, Professor of Pathology, Medical Department, University of Texas, for his valuable and indispensable cooperation in supervising the preparation of the specimens and their study, without which this investigation would have been seriously handicapped. Our discussions have been most helpful, and his opinion on the many sections has aided in making this study as nearly complete as is possible. To Dr. T. L. Terry, Dr.

Gibbs Milliken and Dr. W. Hill of the Department of Pathology, University of Texas, I desire to express thanks and appreciation for assistance in the study of microscopic sections, preparation of some of the drawings and photomicrographs, and, lastly, to Miss Sophie Bachhammer ot Professor Nager's Clinic, University of Zurich, Switzerland, for the excellent colored drawing from a microscopic section.

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TABLE I.

BECK INSTRUMENT

	Muscle	Complete Removal	Chronic Tonsillitis	Abscesses	Lymphoid Hyperplasia	Mucous Glands	Tuberculosis	Miscellaneous
1	Trace	+	Peritonsillitis	—	—	+	—	Perivascular lymphocytes in capsule
2	+	+	Interstitial	—	—	+	—	
3	+	+	Lacunar	—	—	+	—	
4	None	?	No change	—	+	+	—	
5	None	—	No change	—	+	—	—	
6	Trace	+	Interstitial	—	—	—	—	Organized thrombus in capsule
7	Trace	+	Peritonsillitis	—	—	—	+	
8	+	+	Lacunar	—	—	+	—	
9	+	+	Interstitial	—	+	+	—	
10	+	+	Lacunar	—	—	—	—	
11	+	?	Lacunar	—	—	+	—	Plasma cells
12	+	+	Interstitial	—	—	+	—	Perivascular lymphocytes in capsule
13	+	+	No change	—	+	—	—	
14	Trace	—	Interstitial	+	—	+	—	
15	+	+	Peritonsillitis	—	—	+	—	
16	Trace	+	Lacunar	—	+	—	—	
17	Trace	+	Lacunar	—	—	—	—	
18	+	+	Interstitial	—	—	—	—	
19	None	+	No change	—	—	—	—	
20	+	+	Lacunar	+	+	+	—	
21	+	+	Lacunar	—	—	—	—	
22	+	+	Lacunar	—	—	—	—	
23	+	+	Peritonsillitis	—	—	—	—	Perivascular lymphocytes in capsule
24	Trace	—	Lacunar	—	—	—	—	

TABLE II

SLUDER INSTRUMENT

	Muscle	Complete Removal	Chronic Tonsillitis	Abscesses	Lymphoid Hyperplasia	Mucous Glands	Tuber-culosis	Miscellaneous
1	Trace	+	Lacunar	—	—	+	—	Eosinophiles
2	Trace	—	Interstitial	—	—	+	—	
3	+	—	Lacunar	—	—	+	—	
4	+	+	No change	—	—	+	—	
5	+	+	Peritonsillitis	—	—	+	—	Perivascular lymphocytes in capsule
6	Trace	+	Interstitial	+	—	+	—	
7	+	+	Lacunar	—	—	+	—	
8	+	+	No change	—	—	—	—	
9	Trace	+	No change	—	+	+	—	
10	+	+	Lacunar	—	—	—	—	
11	+	+	Lacunar	—	—	+	—	
12	+	+	Interstitial	—	—	+	—	
13	+	+	Lacunar	—	—	+	—	
14	+	+	Lacunar	—	+	+	—	
15	+	+	No change	—	+	—	—	
16	None	—		+	—	+	—	
17	+	+	Lacunar	—	—	+	—	
18	Trace	+	Lacunar	—	—	+	—	
19	+	+		+	—	+	—	
20	Trace	+	Lacunar	—	+	—	—	
21	+	+	Peritonsillitis	—	—	+	—	
22	+	+	No change	—	+	+	—	
23	Trace	+		+	—	—	—	
24	+	+	Interstitial	—	—	—	—	

TABLE III

LaFORCE INSTRUMENT

	Muscle	Complete Removal	Chronic Tonsillitis	Abscesses	Lymphoid Hyperplasia	Mucous Glands	Tuber-culosis	Miscellaneous
1	+	+	Interstitial	—	—	—	+	
2	+	+	Interstitial	—	—	+	—	Eosinophiles
3	+	?	No change	—	+	—	—	
4	+	+	Lacunar	—	—	+	—	
5	+	+	Peritonsillitis	—	—	—	—	Thickened vessel walls
6	None	+	No change	—	+	+	—	
7	Trace	—	Interstitial	—	—	+	+	
8	+	+	Lacunar	—	—	+	—	
9	+	+	Lacunar	+	—	—	—	
10	+	+	Lacunar	—	+	—	—	
11	Trace	+	Lacunar	—	—	+	—	
12	None	—	Lacunar	—	—	—	—	
13	+	?	Lacunar	—	—	+	—	
14	+	+	No change	—	+	+	—	
15	+	+	Lacunar	—	—	+	—	
16	+	+	Lacunar	—	—	—	—	
17	+	+	Lacunar	—	—	+	—	
18	Trace	?	No change	—	+	+	—	
19	+	+	Interstitial	—	—	—	—	
20	Trace	+	Peritonsillitis	—	—	—	—	
21	+	+	Interstitial	—	—	+	—	
22	+	+		+	—	—	—	
23	+	+		+	—	—	—	
24	+	+	Interstitial	—	—	—	—	

TABLE IV

MEDING INSTRUMENT

	Muscle	Complete Removal	Chronic Tonsillitis	Abscesses	Lymphoid Hyperplasia	Mucous Glands	Tuber-culosis	Miscellaneous
1	+	—	Lacunar	—	+	+	—	
2	+	+	Lacunar	—	+	+	—	
3	None	+	Lacunar	—	—	+	—	
4	+	+	Lacunar	—	—	+	—	
5	None	—	Interstitial	—	—	+	—	
6	+	—	Interstitial	+	—	+	—	
7	+	+	Peritonsillitis	—	—	+	—	Eosinophiles
8	+	+	Interstitial	—	—	—	—	
9	+	+		+	—	+	—	
10	None	+	Unchanged	—	—	—	—	
11	None	—	Lacunar	+	—	—	—	
12	None	—	Unchanged	—	+	—	—	
13	+	+	Lacunar	—	—	—	—	
14	+	+	Lacunar	—	—	+	—	
15	+	+	Lacunar	—	—	+	—	
16	+	+	Interstitial	—	—	—	+	
17	+	+	Lacunar	—	—	—	—	
18	Trace	+	Unchanged	—	+	—	—	
19	None	—	Lacunar	—	—	—	—	
20	+	—	Peritonsillitis	—	—	—	+	Eosinophiles
21	+	—	Interstitial	—	—	+	—	
22	Trace	+	Unchanged	—	+	—	—	
23	+	+	Lacunar	—	—	—	—	
24	+	+	Lacunar	—	—	—	—	

TABLE V

DISSECTION METHOD

	Muscle	Complete Removal	Chronic Tonsillitis	Abscesses	Lymphoid Hyperplasia	Mucous Glands	Tuber-culosis	Miscellaneous
1	+	+	Lacunar	—	—	—	—	
2	+	+	Lacunar	—	—	—	—	
3	Trace	+	Lacunar	—	—	—	—	
4	+	+	Lacunar	—	—	—	—	
5	None	—		+	+	—	—	
6	+	+	Lacunar	—	—	+	—	
7	+	+	Lacunar	—	—	—	—	
8	Trace	+	Interstitial	—	—	—	+	Tonsillolith
9	+	+	Peritonsillitis	+	—	—	—	
10	+	+	Lacunar	—	—	—	—	
11	+	+	No change	—	+	—	—	
12	Trace	?	Interstitial	—	—	—	—	
13	Trace	+	No change	—	+	+	—	
14	Trace	+	Interstitial	—	—	—	—	
15	+	+	Lacunar	—	—	—	—	
16	+	+	Lacunar	—	+	+	—	
17	+	+	Interstitial	—	—	+	—	
18	None	+	Peritonsillitis	—	—	—	—	
19	Trace	+		+	—	—	—	
20	None	+	Lacunar	—	—	+	—	
21	Trace	+	No change	—	+	—	—	
22	+	+	Interstitial	—	—	—	—	
23	Trace	+	Interstitial	—	+	—	—	Cartilage in capsule
24	Trace	+	Lacunar	—	+	—	—	

TABLE VI

BAUM INSTRUMENT

	Muscle	Complete Removal	Chronic Tonsillitis	Abscesses	Lymphoid Hyperplasia	Mucous Glands	Tuber-culosis
1	Trace	+		+	—	+	—
2	Trace	+	Lacunar	—	+	+	—
3	Trace	+	Interstitial	—	—	—	—
4	None	—	Lacunar	—	—	+	—
5	None	?		+	—	+	—
6	Trace	?	Lacunar	—	—	—	—
7	+	+	No change	—	+	+	—
8	+	+	Interstitial	—	—	—	+
9	+	+	Peritonsillitis	—	—	—	—
10	+	?	No change	—	+	—	—
11	Trace	+	Lacunar	—	—	+	—
12	+	+	Lacunar	—	+	+	—
13	Trace	—	Interstitial	—	—	+	—
14	Trace	—	Interstitial	—	—	—	—
15	Trace	+	Peritonsillitis	—	+	—	+
16	None	+	Lacunar	—	—	—	—
17	Trace	+	Lacunar	—	—	+	—
18	+	+	No change	—	+	+	—
19	+	+		+	—	+	—
20	None	—	Lacunar	—	—	+	—
21	None	+	Lacunar	—	+	+	—
22	+	+	Lacunar	—	—	—	—
23	+	—	Interstitial	+	—	—	—
24	+	+	Lacunar	—	+	—	—

TABLE VII

DISSECTION METHOD

	Muscle	Complete Removal	Chronic Tonsillitis	Abscesses	Lymphoid Hyperplasia	Mucous Glands	Tuber-culosis
1	+	+	Lacunar	—	—	—	—
2	+	+	Lacunar	—	—	+	—
3	+	+	Interstitial	—	+	+	—
4	+	+	Unchanged	—	+	—	—
5	+	+	Lacunar	—	—	+	—
6	+	+		+	—	+	—
7	Trace	—	Unchanged	—	+	—	—
8	+	—	Lacunar	—	—	+	—
9	Trace	+	Lacunar	—	—	+	—
10	+	+	Lacunar	—	—	+	—
11	+	+	No change	—	+	—	—
12	+	+	No change	—	+	—	—
13	+	+	No change	—	+	+	—
14	+	+	Interstitial	+	—	+	—
15	+	+		+	—	+	—
16	+	+	No change	—	+	+	—
17	+	+	Lacunar	—	—	+	—
18	None	+	No change	—	+	+	—
19	Trace	+	Lacunar	—	—	—	—
20	+	+	Interstitial	—	—	—	—
21	None	—	Interstitial	—	—	—	—
22	+	+	Interstitial	—	—	—	—
23	+	+	No change	—	+	—	—
24	Trace	+	Peritonsillitis	—	—	—	—

TABLE VIII

SEARCY INSTRUMENT

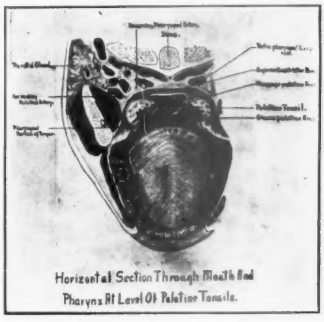
Muscle	Complete Removal	Chronic Tonsillitis	Abscesses	Lymphoid Hyperplasia	Mucous Glands	Tuber-culosis	Miscellaneous
+	+	Lacunar	—	+	+	—	Perivascular lymphocytes in capsule
Trace	—	Lacunar	—	—	+	—	
+	+	Interstitial	—	—	+	—	
+	+	Interstitial	—	—	+	—	

No. 1.

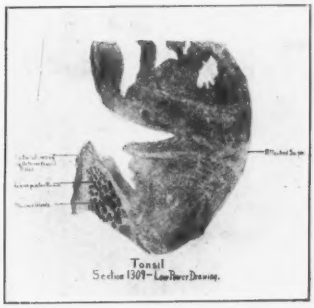
Vertical section including base of tongue from cadaver. Showing relation of tonsil to adjacent muscle tissue with irregularity of aponeurosis and connective tissue. The "so called true tonsil capsule" does not exist—low power.

No. 2.

High power magnification of area of Fig. 1, showing distribution of connective tissue and aponeurosis, and relation to adjacent muscle tissue. From this illustration one can appreciate the obstacles to be encountered in the accomplishment of the "so called capsule splitting."

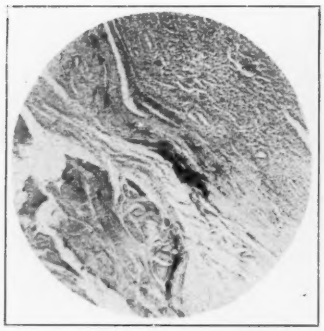


No. 3.

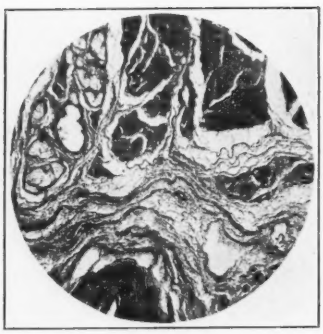


No. 4.

Showing large amount of muscle tissue removed from anterior pillar at time of operation. Removal of muscle tissue is better understood in this section by referring to the illustration of the "Horizontal Section through Mouth and Pharynx at Level of Palatine Tonsils." (Cunningham.)



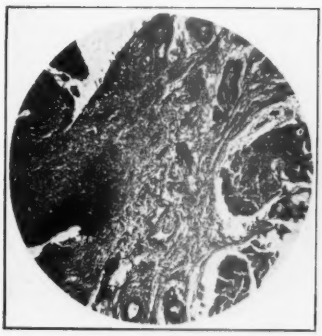
1370.
Beck Instrument.
Muscle and tonsillar tissue.



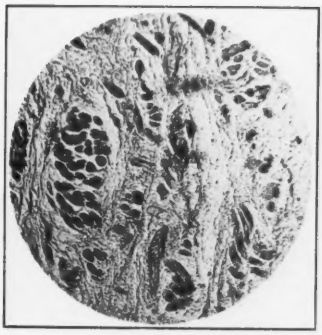
1371-A
Muscle and Epithelium
Anterior Pillar.
Beck Instrument.

1371-B.
Large blood vessel surrounded by muscle. Beck Instrument.

1371-C
Showing large blood vessel sometimes present in the muscles removed. Beck Instrument.



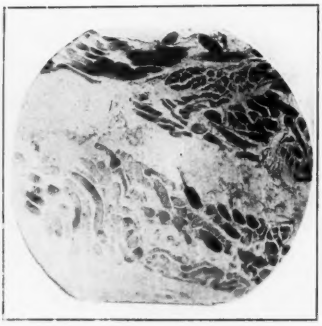
1386.
Muscle, epithelium and mucous glands. From anterior pillar. Low power Sluder Instrument.



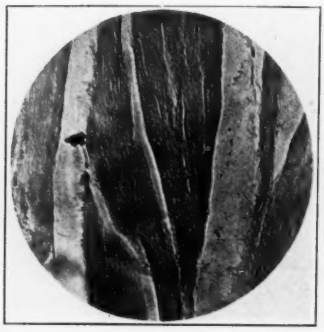
1392.
Muscle. Sluder Instrument.

1395.
Muscle and surface Epithelium.
Sluder Instrument.

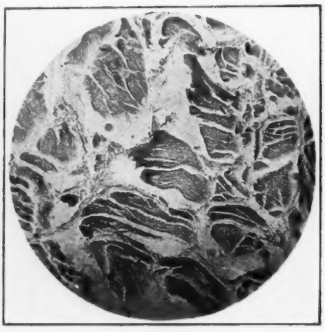
1438-A.
Muscle and tonsillar tissue. Low Power Meding Instrument.



1438-B.
Muscle removed with tonsil. Meding Instrument.



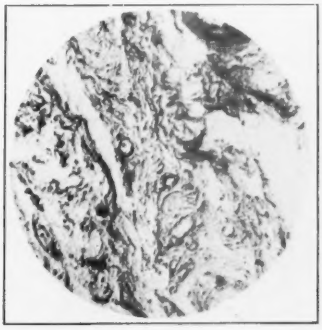
1454-A.
Muscle (longitudinal section). LaForce Instrument.



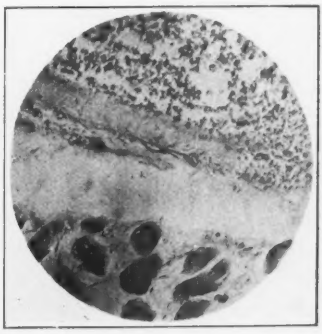
1454-B.
Muscle removed with tonsil. LaForce Instrument.

1494.
Muscle and tonsillar tissue.
Baum Instrument.

1495.
Muscle and hyalinized fibrous tissue. Baum Instrument.



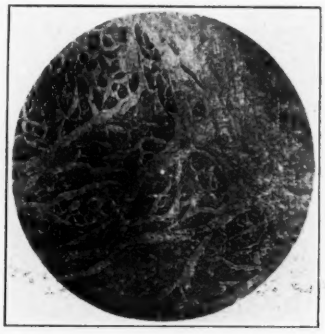
1519-A.
Muscle and tonsillar tissue.
Dissection method.



1519-B.
Muscle and tonsillar tissue with hemorrhage in muscle tissue. Dissection method.

1519-C.
Muscle and fibrous tissue capsule of thickened peritonsillar change. Dissection method.

1519-D
Muscle removed with tonsil.
Dissection method.



1531
Muscle and epithelium.
Searcy Instrument.

IV.

PHOTOGRAPHY OF THE LARYNX.*

By Louis H. Clerf, M. D.,

Philadelphia.

Historical.—Over forty years have elapsed since Mr. Lennox Browne, appearing before the British Medical Association, read a paper on "Photography of the Larynx and Soft Palate," and presented some excellent photographs of the larynx of Mr. Emil Behnke. His work, although limited in its scope, successfully demonstrated a new method of securing records of the larynx which would be accurate and permanent. The progress made in the field of laryngeal photography since that presentation has greatly simplified the procedure. In spite of the facility with which it may be used, the relative infrequency of its application warrants the belief that this form of recording has not received the attention it merits.

The application of the art of photography to medicine presented vast possibilities for the production of medical records of an exact and permanent nature. Such records are of great value in that they often aid in the study of the physiology and pathology of a part or organ; they enable one to provide true illustration for medical histories of patients or for case records to be reported; comparative studies can be made at one's leisure and often they are of great service in teaching. Heretofore drawings had been necessary for purposes of recording observations and results. These usually are more or less schematic and conventional; at best they are only approximate and vary according to the skill of the artist and his familiarity with the subject he is sketching. Often his personality is reflected in his sketches.

Marked advances have been made in the photography of those regions of the body which are readily accessible to the camera, and as a result this method of making records is now

*From the Bronchoscopic Clinic, Jefferson Hospital, Philadelphia, Pa.

commonly utilized. The larynx has not shared equally in this progress. This is due in great part to its relative inaccessibility and the difficulties encountered in its illumination.

On reviewing the literature on photography of the larynx, one is impressed with the fact that but few laryngologists have manifested interest in this subject and that its application has been exceedingly limited. Czermak, who in 1860 made some investigations in photography of the larynx, was the first worker in this field. He was followed by Solis Cohen, Elsberg and others, who also made unsuccessful efforts which, however, were not futile, since they served to stimulate others.

After prolonged and patient effort with the facilities at his command, Mr. Browne (1883) finally obtained results which were gratifying; he was able successfully to photograph the larynx of one subject. However, he did not foresee any future for the general application of photography in laryngology, nor did he believe that the difficulties which so beset his labors could be overcome. It may not be amiss to quote from his paper:

"I do not anticipate that photography of the larynx can be extended beyond the boundary of physiology. To expect photographs from life of pathologic conditions is plainly unreasonable, since those we have seen could only have been obtained by elaborate and costly machinery, and, above all, from a subject possessed of unusual, indeed, in my experience, of unequaled knowledge of what was our goal, and skill and endurance necessary for its attainment."

These prophetic remarks seem to have been handed down and appear to be entertained generally by the laryngologists of today.

In August of 1884, about one year after Browne's presentation, Thomas R. French read before the Section of Laryngology, International Medical Congress, at Copenhagen, a paper "On a Perfected Method of Photography of the Larynx," and presented two portfolios of photographs of normal and diseased larynges and of the posterior nares. These photographs are truly remarkable and are the end results of an enormous expenditure of time and energy as well as infinite patience and persistence. He began his work in 1882 and, during the

two years that followed, experiments were made with every type of camera and the various forms of illumination. With the assistance of Mr. George Brainerd, he finally perfected a satisfactory camera and devised a method to utilize the rays of the sun for illumination of the interior of the larynx. All the means of artificial illumination at that time were found inadequate. Within the space of one short year after Browne's disheartening comments, an enormous impetus was given to this interesting subject by Dr. French. His success is well expressed in his paper, for he states:

"Our object has been not only to obtain good photographs but to devise an easy method by which any expert laryngoscopist can photograph the larynges of his patients in everyday practice. This we have accomplished. Not only have we succeeded in devising a method for photographing the larynges of those accustomed to the presence of the mirror in the fauces, but . . . . of those upon whom the laryngoscopic mirror was used for the first time and who were ignorant of the object of the procedure."

Apparatus Devised by French.—The apparatus which he devised consisted of (1) an instrument designated a sunlight concentrator, (2) a perforated forehead reflector and (3) a camera with laryngeal mirror attached. The sunlight concentrator consists of a hollow truncated cone of metal fitted with suitable lenses to concentrate the sun's rays and provide a powerful light which is practically without heat. This apparatus is fastened to a window frame by means of a rod equipped with a universal joint upon which it is mounted. The camera is a small box with hinged back, which permits the introductoin of a ground glass or plate holder. On the front of the camera is a telescopic tube which, at its distal end, is fitted with the lens system. A drop shutter placed immediately behind the telescopic tube is released by means of a key on the front of the camera. There is a hollow brass tube to the right side of the lens telescope, into which the handle of the laryngeal mirror is inserted and secured by a thumbscrew.

As shown in Fig. 1, the apparatus is in position to take a photograph of the larynx. The rays of the sun passing through the concentrator (A) are received on the concave reflector (B) and reflected through the patient's mouth on

the laryngeal mirror, which in turn reflects the light rays into the cavity of the larynx. The image of the larynx, now illuminated, is received by the laryngeal mirror, reflected into the lens of the camera and a photographic image results. This is identical with the ordinary indirect examination of the larynx except that the examiner's eye is replaced by the camera and, instead of receiving a visual impression of the larynx, the image is recorded on a sensitive photographic plate and can be preserved for subsequent study.

Sunlight as a source of illumination proved disadvantageous, because it limited one's activities to several hours daily and then only when the sun shone brightly. In addition, the varying intensity of the sunlight influenced the success of the exposures; also, patients were not always available when the sun shone brightly. To overcome these disadvantages French devised a means of utilizing the arc light as a source of illumination, with excellent results.

Other workers experimented along lines similar to these set forth by French, but little further progress was made until 1919, when Garel, of Lyons, devised an apparatus which gave us stereoscopic images of the larynx. It is a most ingenious device and the stereograms, when viewed stereoscopically, gave a perspective of the larynx that is indeed wonderful to behold.

Apparatus Devised by Garel.—The apparatus consists of a small rectangular stereoscopic chamber (A, Fig. 2), fitted above with a curtain shutter having a lock and release attachment (B), and so constructed at the bottom (C) as to receive either the ground glass or plate holders (D). Surmounting this is a small cubicle (E), which holds the two objectives and a prism which reflects rays of light at a right angle. A prism meniscus has been added to the prism at a point corresponding to the line of separation of the two objectives. This neutralizes the prism and serves as a view finder (H). On the right is a hollow tube (I), which receives the shank of the laryngeal mirror.

A small tungsten lamp enclosed in a metal telescopic cylinder (K) and fitted with a strong condensing lens is fastened to the left side with a ball and socket joint. The ordinary illumination of the tungsten lamp is insufficient for photograph-

ing the larynx. To obtain sufficient light the lamp must be overilluminated during the moment of exposure. To permit this a resistance coil (L) is used. This is controlled by a foot pedal (M). The speed of the shutter varies from 1/15 to less than 1/60 of a second. It may be released by the left hand of the operator, and should the left hand be occupied in holding the patient's tongue, then a special shutter release (O) may be fastened to the foot pedal and operated with the toe. After a little practice one can press down the foot pedal to overilluminate the lamp, and then, with the same movement of the foot, immediately release the shutter.

Technic.—It has been my privilege to use the stereoscopic laryngeal camera devised and perfected by Garel. Not being conversant with laryngeal photography in any of its phases and without claiming the qualifications of the "expert laryngologist" referred to by French in his original paper, I began to photograph the larynx. Garel recommends that a beginner practice on professional singers, for they are usually able to make a good exposure of the larynx. Because of my inability to secure the services of such a personage, I was content to photograph the larynges of the patients as they came to the clinic. As a result, my early efforts were very discouraging. However, I soon learned to overcome many of the simple errors and, by closely following the excellent instruction of Garel, I was able to obtain good photographs in a fair proportion of the patients examined.

The accompanying stereograms represent some of the results that I have obtained in cases as they appeared in the Bronchoscopic Clinic. To obtain the best results, these illustrations should be viewed with a stereoscope. A satisfactory print of the larynx, when studied with the stereoscope, gives a better idea of its depth than is obtained when the larynx is examined by the indirect method. A majority of the patients examined were quite tolerant to the laryngeal mirror. Four exposures are taken at one sitting and require but a few minutes. Patients subjected to this suffer no greater inconvenience than that experienced when examined with the laryngeal mirror, the exposures being practically instantaneous.

Illumination of Larynx.—It must be remembered that not every larynx is capable of being photographed. There are

many difficulties which must be overcome. The illumination of the larynx is not always easy. One side is usually well lighted, while the other side is so poorly illuminated that no impression is made on the photographic plate. This occurs because the source of light is placed laterally on the left side of the camera. The rays of light, projected through the right side of the subject's open mouth upon the laryngeal mirror, are reflected obliquely to the left side, and the larynx is but partially illuminated. This can be overcome by bending the mirror obliquely in its transverse diameter so that it will face a point almost midway between the source of light and the lens aperture of the camera. It is necessary to move the telescopic cylinder containing the condensing lens, towards or away from the tungsten lamp, to obtain the proper focus of the luminous rays. This circle of rays should not exceed the diameter of the shadow cast by the laryngeal mirror. In addition, the lamp should be placed in a position which will permit the luminous rays to center on the left half of the mirror, slightly below its transverse diameter.

Theoretically a light placed between the objectives should give the best results, because the rays of light would then be more nearly parallel to the reflected image of the larynx. Garel, after experimenting with the light in that position, states (personal communication) that it is impractical, as the epiglottis casts a shadow and permits illumination of but a part of the larynx.

Focusing.—The camera has a fixed focus. This I have estimated to be approximately 23 centimeters from the front of the prism. The depth of the average larynx is about 3 centimeters, therefore the handle of the mirror must be so placed in the brass tube that the central portion of the mirror is about 20 centimeters from the front of the prism. This, of course, is not a fixed distance and must be determined for each case. Having determined the position for the average case, the mirror handle can be marked at this point and additional exposures made with the mirror handle moved one or two millimeters in front or behind this mark. In order to give greater focal depth to the objectives, I have added a diaphragm to each lens, which has cut it down about 25 per cent. This necessitates a proportionate prolongation of the time of

exposure, but in certain cases which are accustomed to the mirror, this can be done without any loss of detail or blurring. Because of the position of the ground glass it is very difficult to determine whether the larynx is in focus. It is impossible for the operator to obtain a direct view of the ground glass. An assistant can view it, but the position he must assume is a very awkward one. A more convenient method is to suspend a mirror below the ground glass at an angle so that the operator, by looking downward from his position at the view finder, can see the reflected images.

These are some of the many sources of difficulty encountered which, to the beginner, seem impossible to overcome. With a moderate amount of practice upon a tolerant subject, these minor difficulties will be readily overcome and but a few of the fundamental problems will be left for solution.

When one recalls the early attempts made in the work and compares the results of that day with those of the present time, one is impressed with the progress that has been made. There are many problems still unsolved. With the constant improvement being made in electric lighting one can hope for a more intense light which will permit the use of color filters and which can be so placed that it will better illuminate the larynx. In order accurately to determine the action of the vocal cords during phonation and in singing, it will be necessary to provide an apparatus which, constructed on the principle of the pharyngoscope, can be used when the tongue and epiglottis enjoy their normal relative position and are unhampered in their movements. I am at present at work on these problems and hope to add a mite to the wonderful results obtained by the pioneers in this field.

Photography of the larynx is not easy if we lack a knowledge either of photography or of the larynx. To attain proficiency one must expend time and energy and cultivate the virtues of patience and perseverance. When once understood, it is easy of application, and successful results are the rule. The possibilities of adding to our knowledge of the larynx, normal or diseased, and the personal satisfaction of obtaining accurate images should be a sufficient compensation for the time spent in developing the necessary technic.

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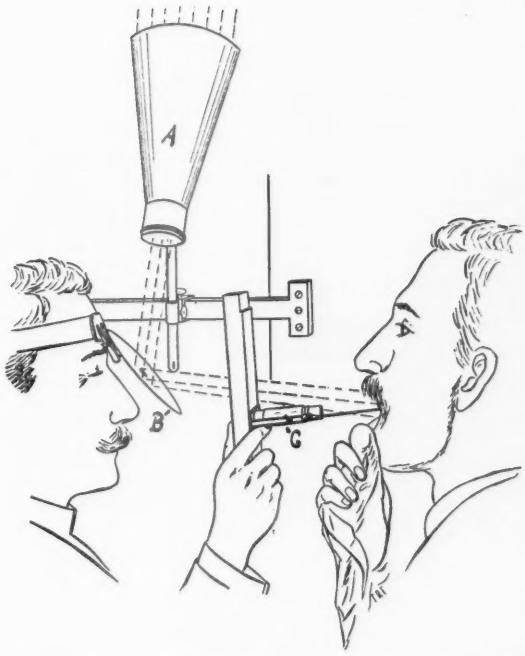


Fig. 1. Sketch showing French's apparatus in position to photograph the larynx. (A) Sunlight concentrator, fastened to the window frame, receives and concentrates the sun's rays. (B) Concave reflector secured to a headband and arranged over the operator's left eye so that the rays received upon it will be reflected into the subject's mouth upon the laryngeal mirror. (C) The camera which received the reflected image of the larynx from the mirror. (Drawn by French.)

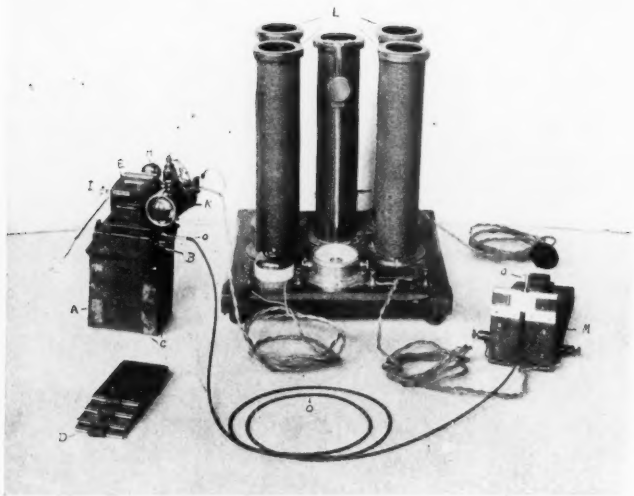


Fig. 2. The perforated stereoscopic apparatus of Garel. (A) The rectangular stereoscopic chamber. (B) Lock and release attachment which control the curtain shutter. (C) Slide which received either the ground glass or plate holder. (D) Plate holders numbered from one to four. These hold the plate which measured 4.5x6 centimeters. (E) Cubicle which contains the two objectives and prism and has extending backward a hollow cylinder. (H) which serves as a view finder. (I) Hollow tube which supports the shank of the mirror. (K) Metal telescopic cylinder containing the lamp and condensing lens. (L) Resistance coil. (M) Foot pedal. (O) Special shutter release which is controlled by the operator's foot. (Garel.)

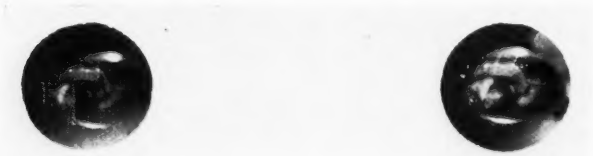
Case No. L. P. 1A. A normal larynx taken on deep inspiration.

Case No. L. P. 1B. A normal larynx taken during phonation.

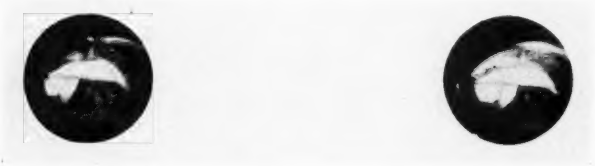
Case No. L. P. 2. Case of feeble laryngeal tensors occurring in a man aged 29 years. History of slight hoarseness in 1918 following a heavy cold. Abduction and adduction normally performed.

Case No. L. P. 3. Vocal nodule with a laryngotracheitis in a woman aged 39 years. Complained of hoarseness which followed loss of voice at a football game three years ago.

Case No. L. P. 4. A fungating mass seen in the posterior commissure. Patient is a woman, aged 40 years. Studies not completed. Probably tuberculous.



Case No. L. P. 5. Granuloma of vocal cord occurring in a young woman of 23 years.

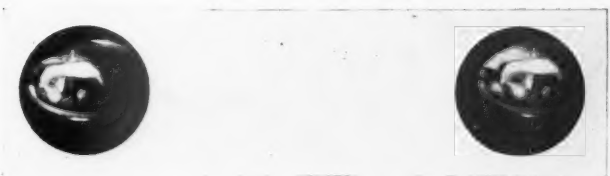


Case No. L. P. 6A. Hemangioma of vocal cord in a woman of 33 years. Hoarseness of one year duration following an attack of bronchitis. Taken on deep inspiration.

Case No. L. P. 6B. Hemangioma of vocal cord in a woman of 33 years. Hoarseness of one year duration following an attack of bronchitis. Taken on phonation. The tumor mass is lying on the adducted cord.

Case No. L. P. 7. Epithelioma of pharynx occurring in a man of 63 years. The right half of the larynx can be seen, the left side being obscured by the new growth.

Case No. L. P. 8. Vocal nodules (singer's nodes) in a woman of 26 years. Larynx in position of phonation.



Case No. L. P. 9A. Bilateral recurrent paralysis in a young woman aged 36 years. Dyspnea since 21 years of age. Voice not hoarse but weak. Following thyroidectomy six years ago, dyspnea increased and there has been some hoarseness. Taken during inspiration.

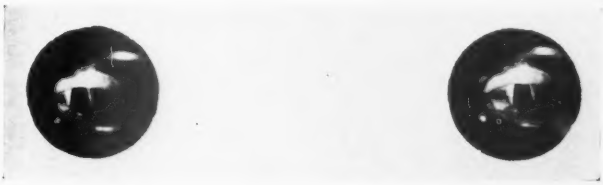
Case No. L. P. 9B. Bilateral recurrent paralysis in a young woman aged 36 years. Dyspnea since 21 years of age. Voice and hoarse but weak. Following thyroidectomy six years ago, dyspnea increased and there has been some hoarseness. Taken durnig phonation.

Case No. L. P. 10. Cicatricial laryngeal stenosis following perichondritis, probably postinfluenzal, in a young man of 21 years. Tracheotomy was necessary to relieve the intense dyspnea. Laryngostomy was done subsequently.

Case No. L. P. 11. Bifid epiglottis, probably congenital, in a man of 69 years.

Case No. L. P. 12. Recurrent laryngeal paralysis occurring in a woman aged 44 years. About 1½ years ago had amputation of breast for cancer. Six weeks ago there was sudden hoarseness, with cough and dysphagia. Mediastinal metastasis.

Case No. L. P. 13. Bilateral recurrent laryngeal paralysis in a woman aged 37 years. Thyroidectomy 3½ years ago. For four weeks preceding operation had "laryngitis" with slight dyspnea. No examination of larynx. After operation there was marked dyspnea with aphonia. Tracheotomy was done later.



Case No. L. P. 14. Unilateral recurrent laryngeal paralysis in a woman aged 32 years. There was an associated cancer of the cervical esophagus.

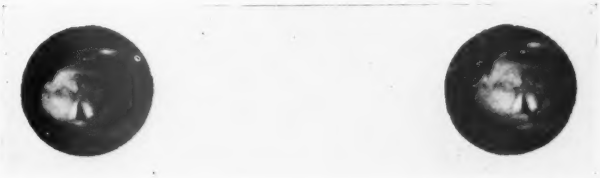
Case No. L. P. 15. Traumatic laryngeal stenosis in a boy aged 14 years.

Case No. L. P. 16. Case of papilloma of larynx in a man of 42 years. History of hoarseness following a cold two years before. Growths removed by direct laryngoscopy. Diagnosis verified by biopsy.

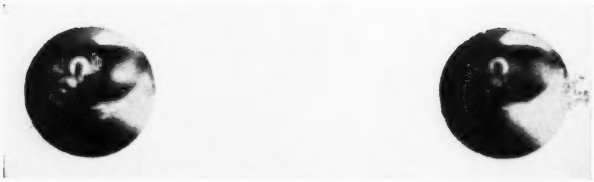
Case No. L. P. 19. Larynx of a young woman, age 23 years. Small granuloma was removed by direct laryngoscopy from the right vocal cord two months ago.

Case No. L. P. 20. Case of cured laryngeal stenosis resulting from high tracheotomy for dyspnea following thyroidectomy. Laryngostomy was done 18 months ago and the case is now ready for closure of the neck.

Case No. L. P. 21A. Woman, aged 52 years, has been hoarse for 15 years. Recently hoarseness has increased and there is dyspnea. There is a large fold on each side which appears to be an eversion of the ventricle. Not typical.



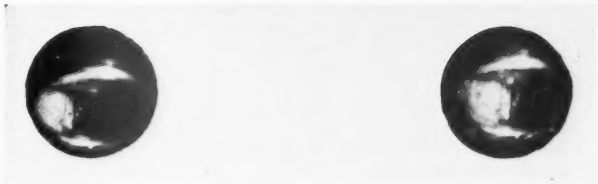
Case No. L. P. 21B. Woman of 52 years. The large folds on each side which seemed to be an eversion of the ventricle were removed by direct laryngoscopy.



Case No. L. P. 22. Very large adult male larynx with infantile epiglottis. Taken during phonation, using a convex mirror. This photograph is one-half actual size.

Case No. L. P. 23. Papilloma of larynx in a woman aged 45 years. Seventeen years ago had a thyrotomy performed in Russia for the removal of a growth. Nodule removed by direct laryngoscopy. Verified by biopsy.

Case No. L. P. 24A. Larynx of man aged 72 years, who had an epithelioma of anterior half of right vocal cord. Operation contraindicated because of patient's condition. Photograph taken 15 months after radium treatment was instituted.



Case No. L. P. 24B. Larynx of man aged 72 years, who had an epithelioma of anterior half of right vocal cord. Operation contra-indicated because of patient's condition. Photograph taken 15 months after radium treatment was instituted. Larynx in position of phonation.

Case No. L. P. 25. Larynx eight months after laryngostomy for cicatricial laryngeal stenosis. The laryngeal airway is ample and the fistula in the neck will be closed by plastic operation.

Case No. L. P. 26. Granuloma of larynx in a man of 63 years. Patient complained of marked hoarseness. Pathologic report was chronic inflammatory tissue.

Case No. L. P. 27. Laryngeal stenosis cured by laryngostomy. History of diphtheria at age of 5 years, with intubation followed later by tracheotomy. Atresia of larynx with buccal voice. Laryngostomy two years ago when 14 years of age.

Case No. L. P. 28. Larynx of young lady of 23 years, who developed pulmonary tuberculosis three years ago, followed by laryngeal tuberculosis one year later. There is a web stenosis of anterior half of larynx with marked infiltration in the interarytenoid space and puffiness of both arytenoids.

Case No. L. P. 29. Fibroangioma of larynx occurring in a woman of 28 years. Hoarseness began three months ago following a cold. Removed by direct laryngoscopy.

Case No. L. P. 30. Marked infiltration of the larynx and subglottic tissues resembling scleroma of the larynx. There is also a similar process involving the posterior choanae. Onset of hoarseness one year ago. Some dyspnea present.

Case No. L. P. 31. Web stenosis of larynx, probably congenital, occurring in a young man of 28 years. Previous history negative.

V.

TREATMENT OF TUBERCULOUS LARYNGITIS, CONSIDERING ESPECIALLY THE USE OF CHAULMOOGRA OIL.*

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The anatomic relationship and easy accessibility of the larynx would seem to greatly facilitate the treatment of its diseases. As far as the treatment of tuberculosis is concerned, however, it cannot be said that rapid progress has been made or brilliant results obtained. This study attempts to explain some of the causes for such a condition and to offer certain suggestions for improvement, devoting considerable attention to the use of chaulmoogra oil as a valuable therapeutic agent. Because of its limited indication, consideration of surgical treatment is purposely omitted.

That the treatment of this disease still remains imperfect is self evident. A few years ago Dr. Robert Levy[1] stated that "no subject in medicine possesses a more extensive or interesting history than tuberculosis, nor can any disease boast of more attempts at solving the problem of its treatment with less permanent, definite and satisfactory results."

Compared with cancer of the larynx no positive diagnostic means, like tissue section and the microscope, are available, and neither is there a therapeutic specific comparable to the X-ray and radium. Compared with laryngeal syphilis, we do not have the Wassermann reaction for diagnosis or the salvarsans for treatment. This lack of progress is possibly largely due to three definite causes, as follows: First,

Inadequacy of Early Diagnosis.

Since early recognition of the disease means so much toward successful treatment, it is not a digression to refer to it

*Candidate's thesis for the American Laryngological, Rhinological and Otological Society.

in some detail here. In that practically all cases occur in persons suffering with pulmonary tuberculosis, early signs of laryngeal complication may first be observed by either the family physician or the tuberculotherapist, often at the patient's home or in the sanitarium. Much has been said as to the ability necessary on the part of the examiner to make an early diagnosis, and volumes have been written concerning the early signs. The wide variation in the amount and stage of the disease, however, found among those referred for treatment, means that some observers discover suggestive signs much earlier than others, and in all tuberculosis the earlier the recognition the better is the chance of cure. Too much is already demanded of the internist in these days of modern medicine to ask that he become a laryngologist. He should, however, be on constant guard for early signs of localization in the larynx just as well as in the abdomen. There are few suffering with tuberculous laryngitis who, at the onset of laryngeal invasion, cannot attribute the hoarseness or "sore throat" to a simple head cold or to some such irritation as smoking, cooling quickly at a dance or to yelling at a football game. The physician, however, must immediately suspect the real cause, and does well right at this time to have an examination by a laryngologist, and his cooperation in managing the case. Mullin[2] is of the opinion that "one of the greatest needs of the modern tuberculosis sanitarium is the service of a competent laryngologist to examine the larynx of every entrant—and in only this way," he states, "can the incipient cases be detected and the advanced ones prevented."

St. Clair Thompson[7] said: "The chief thing in determining the exact condition . . . . . is the skilled eye of the diagnostician."

In addition, frequent routine reexamination of every tuberculous patient is necessary, because of the rather sudden and unexpected onset and because of the great prevalence of this disease. It is universally encountered, being the second most frequent complication of pulmonary tuberculosis. Brown[3] states that "laryngeal tuberculosis occurs in about 25 per cent or more of adults with pulmonary tuberculosis and that in the United States 100,000 people have the disease."

Lockard[4] says that "one-third of all persons with recognizable lesions in the lungs have complicating lesions in the throat; that autopsies upon individuals dead of consumption prove that nearly 50 per cent have tuberculous lesions in the larynx."

Bronfin[5] and Markel[5] say that it "is present in at least 63 per cent of the tuberculous."

L. de Reynier[6] goes farther by saying that "it is rare to find a patient with chronic pulmonary tuberculosis who does not have in the course of his disease a laryngeal localization."

Considering then the prevalence of pulmonary tuberculosis —both known and undetected cases—laryngeal tuberculosis is far from being an unusual disease, and its eradication may be hoped for only when its extremely early recognition becomes universal.

INCURABILITY.

The second great handicap to progress is the idea still existing among many physicians, laryngologists included, that tuberculosis of the larynx is an incurable disease. That this idea still prevails may be hard for many laryngologists to believe, especially those residing in the mountainous states, where the disease is very prevalent, but it is, nevertheless, true. It was recently verified, with some surprise, by the writer, that competent teachers of laryngology in one of our Eastern medical centers, although located where the disease is not very common, were of the opinion that nothing worth while could be done for tuberculosis of the larynx. The same idea was found to exist among those discussing a medical paper in one of our Southern States, it being quite apparent that the participants were not familiar with early signs or with healed larynges, but thought only in terms of advanced and incurable cases. Moreover, the following statement appears in an excellent textbook, just finished by a leading American laryngologist living, however, in a climate that few tuberculous patients would seek:

"The treatment of tuberculous laryngitis is directed primarily toward the focus in the lung, and that is more in the realm of other fields of medicine. The local treatment to the larynx is practically of no curative value, and only palliative

measures have a place in the management of this disease."[8]

Believing that this idea is largely erroneous and that its propagation actually retards progress in the treatment of this malady, the writer has sought to substantiate his own very different ideas by those of other men who likewise treat the disease, obtaining good results. The following few, of many obtainable citations, are offered. L. de Reynier[9] considers laryngeal tuberculosis exceedingly amenable to treatment. He says "the prognosis of laryngeal tuberculosis, except in young infants and cases complicated by syphilis, is relatively benign, since cure is not the exception but the rule."

Lockard[10] speaking of advanced laryngeal tuberculosis, but referring to a surgical procedure, says: "One case of extensive laryngeal and pulmonary involvement operated upon in 1902 lived until 1916, when he died of spinal tuberculosis; a second, with universal laryngeal involvement, second stage pulmonary tuberculosis and tuberculosis of the epididymis, made a perfect recovery, so far as the larynx is concerned, and lived for four years, when he died from extension to the kidneys; a third, who had an Omega shaped epiglottis, infiltration of the right arytenoid, ventricular band and aryepiglottic fold, associated with large cavities in both lungs, and a general ichthyosis, made marvelous improvement after the operation, performed six years ago, and is now a college lecturer."

Dr. Robert Levy[31] says: "The curability of laryngeal tuberculosis is no longer a mooted question, although the means by which the cure is accomplished is still a fruitful source of difference of opinion." He also demonstrated at a recent meeting of the Midwestern Section of the American Laryngological, Rhinological and Otological Society, a most beautiful series of healed larynges in persons at the time leading normal, useful lives and having perfectly normal laryngeal function.

Many similar examples may be seen in our own clientele as well as in that of our colleagues. The literature is rich with abundant proof of the curability of this disease and, even in the early '90's, Keimer[11] recognized the disease as a curable one, and in 1894 wrote a paper entitled "May We Still Speak of the Incurability of Laryngeal Tuberculosis?" Such evi-

dence, together with his own cases, and case reports of others, lead the writer to believe that practically every case of incipient nonulcerative laryngeal tuberculosis is curable; that most cases of the moderately advanced ulcerative type are curable and that some far advanced lesions with much destruction of tissue can be cured. It is to be understood, however, that a healed larynx, like a healed lung, consists largely of scar tissue, is often deformed and distorted and may have diminished function, yet the patient lives, a well man, with a useful larynx.

INDIFFERENCE OF LARYNGOLOGISTS.

The third handicap which retards progress is the indifference of laryngologists in treating this disease, chiefly because many tuberculous persons are considered undesirable patients. The larynx offers a most excellent opportunity for study of living pathology, but is more dangerous to the student than certain other diseased organs, such as the ocular fundus. To a large extent this danger has been overcome in our practice by use of a plate glass shield, suspended between the face of the examiner and the mouth of the patient, and in bed patients by use of a mask about the mouth and nose of the examiner, such as is used by surgeons during operations. Gentle handling of the patient, to induce cooperation on his part, has also worked wonders in preventing the cough. It is sometimes also necessary to diminish pharyngeal reflexes by the spraying of a weak cocain solution, but this procedure is rarely necessary. Mullin[12] says, in offering a reason for this indifference, that laryngologists "do not take an active interest in this disease. Some have told me that they see very little tuberculous laryngitis in their part of the country; but this means only that they are not making the diagnosis. Some are afraid of becoming infected themselves, while examining or treating this condition. Other men have told me that they do not care to treat this disease, but prefer to send the patients to a sanatorium."

Arrowsmith[48] says: "The attitude toward tubercular laryngeal disease of the profession at large, I mean that of both general practitioners and most laryngologists, constitutes one

of the greatest reproaches to which we have ever subjected ourselves in our history as medical men."

The writer believes that when these three conditions are improved, much will have been accomplished in the successful management of this malady, and makes a plea to fellow laryngologists for more interest in the treatment of this widespread disease.

EARLY HISTORY OF TREATMENT.

Referring to the literature on the treatment of laryngeal tuberculosis, beginning with the very earliest publications, one is forcibly struck with the similarity existing between the older and the more recent articles. As early as 1790, 143 years ago, Savremone[13] published a paper on "Therapeutics of Tuberculosis of the Larynx," which is the first authentic one I have been able to find. Antedating this, however, by hundreds of years were certain references to various phases of the disease, but very little if anything was said concerning the treatment. The flow of contributions has gradually increased to the extent that these first 23 years of the twentieth century have produced about the same amount in volume as the total previously produced. Also, it might be stated that with few notable exceptions, a reader would have difficulty in determining whether a given article were a product of the nineteenth or of the present century. This does not, however, necessarily mean to preclude progress. Constant, indeed, has been the search for some new and curative treatment, though questionable have been the results.

Reviewing some of the earliest methods of treatment, the statement is made by Wright[14] that "in 1818 creosote was used in the form of a fumigation of tar, and this might be useful in laryngeal phthisis, but as a rule treatment was regarded as of no avail."

Thus creosote seems to be the first drug used in this connection, so far as the records show. In 1821 F. Siemerling[15] wrote a case report on the "cure of a case of phthisis laryngea through the use of Herring's milk," followed in 1824 by Plasse's[16] article on "Observations of the Usefulness of Mercurial Inunctions in a Case of Laryngeal Phthisis with Suppuration of the Tongue," and again in 1826 by Fischer's[17]

article on the "Premature Expectations of the Therapeutic Value of Herring Milk in Laryngophthisis." In 1836, however, Trousseau[18] indicated the immaturity in the understanding of the disease at that time by the following entitled paper, "Memoire in Reply to These Questions: What does one mean by laryngeal phthisis? What are the organic alterations, the causes, varieties, prognosis? What is the treatment?" In 1842 Neuber[19] wrote on "Laryngophthisis Cured Through Use of Potassium Hydroiodate," while in 1857, Rethis[20] wrote on "The Treatment of Laryngeal Tuberculosis with Calcium Phosphate. From that time on to the present, calcium has been used rather extensively in the treatment of tuberculosis. These early writers, having no knowledge of the pathology or bacteriology of the tubercle, it is reasonable to assume that many of their supposed cases of tuberculosis were in reality lues or cancer. In 1859, Pastor[21] wrote of the "Natural Cure of Laryngeal Phthisis," and in the same year Marotte[22] on the "Successful Use of an Ammoniacal Potion Containing Opium in Two Cases of Laryngeal Phthisis Accompanied by Symptoms of Suffocation and Asphyxia." The opium here mentioned probably embodies the modern idea of the "rest treatment" of tuberculosis, as it likely produced silence as well as relief of pain, and if so, is the first mention of it, to my knowledge, in the literature.

In recent years considerable has been said about a type of "submucous induration." Beck's[8] textbook says: "There is one form of tuberculosis of the larynx that does not show very much on laryngeal examination, in which the pathologic process is a subepithelial infiltration and which is sometimes baffling in a differential diagnosis between lues and tuberculosis."

A similar observation as to pathology was evidently made years ago by Munch,[24] and in 1867 formed the title of his paper, viz., "Chronic Swelling in the Submucous Tissue Which Reaches to the Cartilage Arytenoid; Appearance Similar to Pulmonary Tuberculosis; Recovery Through Cauterization." In 1877, Sawyer[25] published an article on "Nitrate of Silver in Laryngeal Phthisis." In 1878, Cadier's[26] paper appeared on "Laryngeal Phthisis. Treatment of Ulcerations and Oedema of the Larynx by Local Applications of Glycerine

Creosote." Although used sixty years previously, it is only since about this time that creosote has maintained popular prominence in the hands of many treating the disease. In 1879, Sakolowski[27] published his article entitled "Climate in Treatment of Laryngeal Tuberculosis," and since about that decade climate has received much favorable consideration. In 1881, Masini[28] published his paper on "Iodoform in Laryngeal Phthisis," and heralded a drug used in one form or another most extensively until the present day. The following year Schnitzler[29] gave a paper on the "Influence of Iodoform" on the curability of laryngophthisis; followed in 1883 by Semon's[30] paper on "Iodoform as a Local Remedy in Laryngeal Phthisis," and Metzler[31] in the same year wrote on "Iodoform Treatment of Laryngeal Phthisis."

In the year 1885, Herman Krauze[32] advocated and proved the value of lactic acid. This was without doubt the greatest step in that period, and for years following many cures of ulcerative lesions were reported through its use. No drug was more universally employed and none gave better results. Until the present, many laryngologists use lactic acid in all their cases, with much satisfaction, although others have completely discarded it. In 1887, Heryng demonstrated a case "healed by the use of curets and lactic acid."

From 1887 to 1900 numerous papers appeared advocating the use of many different drugs and procedures, but none were of great importance. As illustrative of some of the more unusual of these, one might mention the paper of Campana's[34] in 1893, entitled "Grave Case of Laryngophthisis Treated Successfully With Testicular Liquid." At this period it must be remembered that, regardless of many isolated cures and some individual ideas to the contrary, most medical men considered this an incurable disease. It is then not to be wondered at that the study of a disease commonly considered hopeless had not produced brilliant results. However, the following list of proposed remedies, some of them requiring many trials and eliciting much discussion as to their value, shows that medical minds have not been idle: Herring's milk, mercury, potassium cantharidinate, potassium hydroiodate, calcium phosphate, opium, ammonia, silver nitrate, creolin, napthol, glycerin, creosote, iodoform, iodol, sulphur,

cocain, lactic acid, caffein, menthol, parenchymatous injections, hot air, hydrogen peroxid, camphor, ichthyol, gold, tuberculin, serums, styracol, cultures, Koch's lymph, testicular liquid, glycerophenol lactate, phenolsulforacin, parachlor phenol, bromoform, formaldehyde, guaiacol, protonuclein, orthoform, trichloracetic acid, formalin, argyrol, propesin, anesthesin and scarlet red.

In 1906 Semon[35] made a great step forward by advocating the value of absolute silence. Rest, either complete or partial, seems to be combined with nearly every valuable method of treatment advanced since that time. Is it not possible then that rest is the more important therapeutic measure, credit for healing often being given to some drug or procedure which in reality is of limited or questionable value? Rest is certainly nature's own remedy. Laryngologists residing in the mountainous states, where the influx of tuberculous patients from other localities is continuous, are struck with the number coming because of aphonia. The involvement is so slight that often no lesion can be demonstrated clinically or at postmortem, and yet nature sounds her early warning by making it unpleasant for the larynx to function and cures by the rest thus obtained. There can, in fact, be no doubt that a large majority of spontaneous cures of laryngeal and pulmonary tuberculosis are surely largely the result of rest. Later, nature becomes more insistent to the unheedful, and makes it physically impossible for the larynx or body to function, and then cures the disease by enforced rest. These facts, then, are evidence that rest plays a most prominent rôle in the cure of this disease. Progress of the last two decades is more or less familiar to all and fresh in the memories of many. The perfection of electric cautery by many coworkers and of heliotherapy by Verba[36] and others are certainly valuable contributions.

CHAULMOOGRA OIL.

The most recent therapeutic measure to attract general interest is the use of chaulmoogra oil and its derivatives. Like all new remedies, this drug had its early overenthusiastic supporters, whose claims were too great. Others were more than pessimistic. Now, more than two years' use by several dif-

ferent laryngologists would seem to permit of definite conclusions as to its real value. The writer has, therefore, sought to collect the evidence from those having experience in its clinical use, and to determine with finality therefrom, and from his own experience, whether the drug warrants universal acceptance or should be relegated to the long list of valueless remedies.

Unlike most other remedies, however, chaulmoogra oil therapy is based upon sound scientific investigation. Its chemistry was thoroughly worked out by many capable men,[37] and it was tried experimentally upon animals before it was used in the human larynx. Among the earlier experimental workers were Voegtlin,[38] Smith[38] and Johnson,[38] who reported, in 1921, a most interesting series of animal experiments upon guinea pigs. The following paragraph is quoted from their paper:

"The success reported in leprosy led us to test out the therapeutic value of chaulmoogra oil and its principal components in experimental tuberculosis, for the following reasons: (1) The leprosy and tubercle bacillus belong to the same group of acid-fast bacteria, and (2) the work of Walker and Sweeney seems to indicate that chaulmoogra acid, the specific acid contained in chaulmoogra oil, exerts a powerful action on all acid-fast bacteria, but is inert toward nonacid-fast organisms. Indeed, these authors present figures which show that chaulmoogric acid is 100 times more bactericidal than phenol for acid-fast bacteria, including the tubercle bacillus."

However, their results seemed to warrant the conclusion that experimental animal tuberculosis was not inhibited by chaulmoogra oil.

Kolmer,[39] Davis[39] and Jager[39] studied the action of the drug experimentally, using the bovine strain with similar discouraging results. Undiscouraged, however, by these negative results as to its value, in generalized experimental tuberculosis, laryngologists, stimulated by the findings of Walker[40] and Sweeney[40] as to the bactericidal properties of the oil, began to use it in the tuberculous larynx. It was hoped in chaulmoogra oil, as it had been in scores of former new remedies, that the panacea had been found, an agent which would kill

the tubercle bacilli in the tissue and neutralize its toxins, without being harmful to that tissue.

Dr. R. M. Lukens[41] was one of the pioneers, if not the first, to extensively use the oil in treating tuberculous laryngitis, and in early 1921, he reported results in 60 cases so treated over a period of eight months. He used the intratracheal and intralaryngeal injection method, dropping in, drop by drop, 1 c. c. of 10 per cent or 20 per cent solution in olive oil. His conclusions are as follows:

"1. The chief value of chaulmoogra oil is in the relief of pain and dysphagia.

"2. The relief is continuous, in contradistinction to that produced by cocain.

"3. The treatment is not unpleasant or distressing, is without untoward reactions in the larynx, and can be used without previous cocainization.

"4. Improvement, while not all that could be desired, seems better than that obtained with other drugs."

Drs. Robert A. Peers[42] and Sidney J. Shipman[42] report that same year results in seven cases of far advanced tuberculosis of the lungs and larynx. An effort was made to relieve symptoms only, the patients being too far advanced to hope for more. The laryngeal lesions involved epiglottis, arytenoids and cords. The technic, dosage, etc., was that described by Lukens. Their tabulated results show that in only one of the seven cases was there relief of dysphagia. The other six cases grew steadily worse, but the authors seem rather to feel that this was more the result of progress in the pulmonary lesions than of any detrimental effect the oil might have had on the larynges. Their conclusions, copied from their paper, are as follows:

"1. Intralaryngeal applications of a 10 to 20 per cent solution of chaulmoogra oil in olive oil may relieve the dysphagia of an advanced laryngeal lesion entirely, but such relief has followed its use in only one out of our seven cases. Slight temporary relief occurred in every case.

"2. On the other hand, the general condition of a febrile patient may possibly be unfavorably influenced by such treatment.

"3. Caution should be used in the laryngeal treatment of tuberculosis with chaulmoogra oil until more accurate data are in our possession concerning its local and general effects."

Drs. Frank L. Alloway[43] and James E. Lebensohn[43] of Chicago report 40 cases of tuberculous laryngitis treated with chaulmoogra oil, using principally the technic of Dr. Lukens, except that their patients were treated daily and were classified for study into three groups. Group No. 1 consisted of patients having "distinct but not extreme laryngeal lesions," with symptoms varying from mild hoarseness and slight pain to "definite dysphagia." In this group, symptoms seemed easily controlled oftentimes after only a few treatments. Some, which at first responded slowly, made excellent progress when Lake's pigment was used with the oil. Their second group consisted of those having very slightly physical signs with slight irritative throat symptoms and hoarseness. "These cases often cleared up surprisingly after a few treatments." Group 3 comprised the advanced cases which had been treated with the ordinary methods without benefit. Chaulmoogra oil did not cure them or furnish "any lasting comfort." Their conclusions are as follows:

"Chaulmoogra oil, we believe, should have a definite place in the treatment of laryngeal tuberculosis. It is useful in the majority of cases, though in the treatment of laryngeal tuberculosis as a whole it cannot entirely replace the other forms of medication, such as the cocain-epinephrin spray, orthoform, menthol and Lake's pigment, superior laryngeal nerve blocking, epiglottidectomy, etc.

"Some may be skeptical of the value of chaulmoogra oil, as employed in these cases, because the mechanism of its action is not clear. It may be true that the oil has no specific effect, but its beneficial action can be perhaps adequately explained by its detergent, anesthetic and counterirritant properties. Barwell[44] says: 'Intratracheal oil injections act in the same way (i. e., like steam inhalations and oil nebulæ) in relieving dryness and soreness of the throat, and I have found them very useful in cases of advanced ulceration, reducing inflammation and relieving discomfort.' He employed a 1 per cent solution of menthol in liquid petrolatum, but from both

theoretical considerations and clinical experience it would appear that chaulmoogra oil should act more efficaciously."

There are those not only "skeptical" as to the method of action but also as to the value of the oil itself. Others, too, have feared that real harm might follow its use, and in 1922 Corper[45] and Freed[45] published results of their experiments which showed the action upon lung tissue of intratracheal injection of oils. Chaulmoogra oil in varying strengths was injected into the trachea of rabbits. Conclusions are quoted as follows:

"Chaulmoogra oil and the mixed esters of chaulmoogra oil injected intratracheally into rabbits produce distinct pathologic changes in the lungs, which may range from a profound acute pneumonic consolidation with abscess formation to a proliferative bronchopneumonia, depending on the concentration and the localization of these oils in the lungs. A concentration of these oils, as low as 10 per cent in olive or liquid petrolatum, injected intratracheally, still produces a distinct proliferative bronchopneumonia. Olive oil and liquid petrolatum injected intratracheally into rabbits in small amounts (not to exceed 1 c. c.) are readily aspirated into the finest pulmonary divisions—the alveoli—to be retained for a relatively long time (months), where they can produce a mild type of a proliferative bronchopneumonia. This study verifies and emphasizes previous observations by others indicating that great care is necessary in the introduction of any foreign substances into the lungs normally, and especially in pulmonary diseases. It has been suggested, but scientific proof is lacking, that the fibrotic changes subsequent to the injection of certain foreign substances into diseased foci in the lungs may have a beneficial action on the diseased area (lung abscess or bronchiectasis). It is questionable also whether even local application, as is accomplished by use of the bronchoscope, will insure that the foreign substance may not leave the focus of intended localization to exert a deleterious action on normal lung tissue some distance away. These questions require further detailed study."

Regardless of their ability to produce pulmonary inflammation and destruction in rabbits, by intratracheal injection of

large doses of chaulmoogra and other oils, it would seem from the number of laryngeal cases reported to the present time, without any ill effects in the lungs—even far advanced tuberculous lungs—that the drug may be safely used in treating the human larynx. This idea is verified by Lebensohn,[49] who since his original paper has written a second, reporting 160 additional cases, treated intratracheally with chaulmoogra oil. Especially careful observation followed each injection and led him to conclude that "There is no clinical evidence that such injections judiciouly employed cause any constitutional disturbance, either immediate or remote."

Moreover, during the past months, internists have been injecting chaulmoogra oil intramuscularly and even intravenously in the treatment of pulmonary tuberculosis. While such treatment is as yet too early in its experimental stage to permit worth while conclusions, it surely has possibilities, and intravenous injection without untoward effect would certainly tend to disprove the idea of danger from intratracheal use. Determination of proper intravenous dosage seems as yet unsettled and a difficult problem. Dr. Henry H. Lissner[46] is one of the pioneers, at least in this country, in this field of investigation and reports in detail using chaulmoogra oil in 25 such cases, covering a period of from three to twelve months. It was used intramuscularly, orally and intravenously —the last being the method of choice, and in from 0.5 to 1 c. c. doses. His cases showed no ill effects at any time and some showed improvement in cough, a diminished bacilli content in sputum, a decrease in lung infiltration and signs, and a general improvement. Two of his cases also had laryngeal tuberculosis and both improved without local laryngeal treatment when he administered chaulmoogra oil intravenously. Lissner[46] also believes that some of the undesirable results reported by others come from using too large doses. He advises 2 to 4 minims once weekly, and never more than 0.5 c. c. as a maximum dose, and that at intervals of two or three weeks. He believes, with these precautions, that esters of chaulmoogra oil are valuable in the treatment of pulmonary tuberculosis and also its complications. In this same article Lissner says that King Phillip Brown[46] considers chaulmoogra oil applied to the larynx the most effective remedy

for laryngeal tuberculosis. Others who have used it, however, are far less optimistic.

CASE REPORTS.

Realizing the limitations of the usual methods of treatment of this disease, and encouraged by the good reports of others, we began using chaulmoogra oil in some of our cases of laryngeal tuberculosis over two years ago. While ours are too few in number (25 at the present time) to permit ironclad conclusions, it is thought that their addition to the others already reported might be helpful. The patients have all been private ones, and all the data desirable has been obtainable and accurate records kept. While it has been impossible in every case so treated to use at the same time the older methods of treatment on a parallel case for a control, an effort has been made to do this, and fairly accurate comparisons have been obtained. All cases have had active coexistent pulmonary tuberculosis and received treatment from an internist during the period of our treatment.

Our cases, too, have been divided into three groups for the purpose of making conclusions more definite and specific. It might not be said, for example, that a remedy which proves quite beneficial for incipient laryngeal tuberculosis is beneficial for advanced cases. An enthusiastic advocate of any remedy might be dealing only with easy, early cases and obtain remarkable results, while another observer might use the same remedy with severer cases, meet with poorer results and condemn a really worth while measure. Medical history shows such to have been the case with many remarkable discoveries, and accounts largely for the diversity of opinion always found on any subject in medical literature. To avoid this pitfall, then, we have divided our patients having tuberculosis of the larynx into the following fairly distinct classes, the division being a combined clinical and pathologic one. Class A includes patients having only vascular changes of the laryngeal mucosa, either anemia or hyperemia, which is usually first observed upon the posterior laryngeal wall; it is chronic in its nature and accounts for early hoarseness or aphonia. It does not respond to treatment for simple laryn-

gitis and always accompanies pulmonary tuberculosis. Class B cases are characterized by chronic inflammation, extending deeper than mucosa, induration being the striking sign often accompanying superficial ulceration. Some pain and impairment of function usually result from the induration and always from ulceration. Class C is characterized by necrosis with marked infiltration. Decreased mobility and pain are always present.

In a general way, the technic, dosage, etc., of Lukens has been followed. The largest group in which we have used chaulmoogra oil is Class A. Since most patients whose disease in the larynx is discovered at this early stage tend to get well of the laryngitis, regardless of the kind of treatment employed, our conclusions are not of much value concerning them. A typical case might be cited. Dr. F., a college professor, came to Colorado several years previously on account of pulmonary tuberculosis, which has improved to the extent of permitting his teaching most of the time, although constantly under the care of a competent internist, who has given him much tuberculin. This was his first throat attack. A few months previously, in the midst of an unusually heavy lecture course, his voice began to fail, the larynx was painful and soon aphonic. His laryngeal mucosa was red and was treated daily in a routine manner, including bed rest and voice rest, by his physician, without improvement. After several weeks he was referred to us, found to be aphonic, and to have the following laryngeal findings: Chronic congestion of laryngeal mucosa in general, but most marked on posterior wall and interarytenoid space. Suggestion of infiltration of posterior wall, but not positive. No ulceration. The treatment, in brief, was instillation of 1 c. c. of chaulmoogra oil into larynx two to three times weekly and complete silence. At the end of three months the patient was discharged with normal larynx. He was seen one year after discharge and had no suggestion of any recurrence, although lecturing to classes every day. There was no change in pulmonary condition and no change in weight during the period of our observation. His condition at present is the same as when discharged by us, there being no return of his trouble, although he lectures to classes daily.

Another, briefly summarized, is the case of Mrs. S. R., who came to Colorado from Missouri six months before consulting us, on account of pulmonary tuberculosis with hemorrhage. Complained of hoarseness and dyspnea for past six weeks. Examination showed rather marked chronic congestion of posterior laryngeal wall, of posterior end of right vocal cord, with slight congestion of right aryepiglottic fold and right arytenoid. No induration or ulceration. Treatment was complete silence, rest in bed, and chaulmoogra oil, used one to three times weekly. Improvement in laryngeal condition was continuous and cure apparently complete, patient being discharged and returning to her home in eight months, with no recurrence up to the present time.

The question as to whether or not this class of patients really have tuberculosis of the larynx or have a simple non-tuberculous laryngitis will, of course, be raised. Since no autopsy has, to our knowledge, actually proven the point, and doubtless could not do so, the question remains one of personal opinion.

Carmody[30] says: "Under the term laryngeal tuberculosis may be considered anything from slight hyperemia or anemia to complete destruction of the larynx in the patient afflicted with pulmonary tuberculosis."

Our experience, however, in watching and treating such cases in past years, together with that of colleagues, leads us to believe that every case of tuberculosis of the larynx begins insidiously and passes through the stage illustrated by cases just cited. It is analogous to the early invasion of the lungs, in which often a rather voluminous hemorrhage comes like a cloud from a clear sky, without any other symptom, and with no sign elicitable by present known diagnostic methods. We, therefore, believe that any person suffering with recognizable pulmonary tuberculosis who develops even only a congestion in the larynx, resistant to treatment for simple laryngitis, persisting for a considerable time, and lues, malignancy, tonsillitis, pharyngitis and sinusitis being excluded, does most likely have tuberculosis of the larynx and should be managed accordingly. Such cases comprise our largest group and all persons in Group A have gotten well.

Such satisfactory results were not obtained on all Class A patients before using chaulmoogra oil, as some progressed into Class B and C. However, since beginning to use chaulmoogra oil no Class A case treated by the older methods has progressed into Class B, as any person used as a control who did not do well under the old method was immediately treated with chaulmoogra oil, the patients all being private ones and their welfare considered more important than the experiment. Each time the treatment was so changed the signs of improvement followed quickly and were constant. No patient being treated with chaulmoogra oil was ever changed back to the older methods, his own welfare contraindicating it, in our judgment.

Class B patients—those having induration or ulceration added to chronic vascular changes—are briefly illustrated by the following:

L. M. S. came from Texas, July 23, 1923. History of husky voice for several months following influenza. Had lost several pounds weight, felt run down and tired out. Diagnosis of early pulmonary tuberculosis by internist. Larynx, right arytenoid thickened with chronically congested mucosa. Each vocal cord had a "fuzzy edge," and there was a small nodule on the free border of the left cord at anterior end of posterior third. Diagnosis: Laryngeal tuberculosis, bilateral. Treatment: Chaulmoogra oil dropped into larynx one to three times weekly. Complete voice rest, 18 hours' daily bed rest. After two months patient showed marked improvement, a gain of 18 pounds in weight and disappearance of laryngeal symptoms and signs. At the present, October 23, 1923, after three months' treatment, larynx appears normal with no aphonia, although treatments have been advised, at less frequent intervals, for a few weeks longer.

Mr. B. came to Colorado several years ago with moderately advanced pulmonary tuberculosis, from which he made a good recovery. Following some unusually hard physical labor, he developed a marked dysphonia, and after this had persisted for several weeks he came to us on February 21, 1922. The whole posterior wall of the larynx was red and thickened (submucosal infiltration), as was the left vocal cord. No ulceration or exudate. Chaulmoogra oil was instilled two

or three times weekly during the following two months and occasionally throughout that summer. All signs and hoarseness disappeared within a few months' time. More than a year later, viz., August 1, 1923, patient was reexamined at our request, and the larynx was normal with a rich deep voice. His general health was good, having reached 200 pounds in weight, and he was driving a milk wagon, the daily work beginning at 4 a. m.

J. E. J. came first on November 26, 1921. Aphonia had been complete for several weeks following hoarseness of three years' duration. Sputum was positive to tubercle bacilli at five examinations, and he had suffered two pulmonary hemorrhages. The laryngeal picture was one of intense chronic induration of arytenoids, interarytenoid space, ventricular bands, vocal cords and posterior laryngeal wall. The mucosa was red and thick but intact. He was treated with chaulmoogra oil and absolute silence (and advised complete bed rest which he refused), two or three times weekly from November 26, 1921, to February 16, 1922, at which time he moved away with larynx normal and voice perfect. A laryngologist in the town to which he moved reports later that patient's larynx was then still normal.

Another in this class was Mr. R. P. P., who came first on July 31, 1922, with a positive diagnosis of pulmonary tuberculosis. History states that for the past three weeks throat had been painful continuously and voice very husky. The interarytenoid space and posterior laryngeal wall showed marked infiltration. The vocal cords were congested, the arytenoids and ventricular bands quite red, arytenoids somewhat thickened but no ulceration. Chaulmoogra oil was instilled one to three times weekly from July 31 to October 2, 1922, at which time his larynx was apparently normal and voice perfect. He had gained weight from 148 to 180 pounds and was discharged by us and his internist, and permitted to return to his home in Nebraska and has had no recurrence. This was a Class B patient, although his laryngeal symptoms had existed for only three weeks.

Mrs. W. E. B. came from Iowa in October, 1922, with the following history: A year and a half previously patient became hoarse at a dance, which preceded by a few weeks a

pulmonary hemorrhage, and by a few more weeks finding of tubercle bacilli in sputum. Had been hoarse or aphonic until the present. Temperature now 100° (some days reaches 102°), pulse 148, blood pressure 162/98, weight 140 pounds. Laryngeal mucosa was everywhere slightly congested and greatly thickened but intact. Arytenoids were greatly swollen, as was posterior laryngeal wall. Absolute voice rest was prescribed by us and bed rest, 24 hours daily, by internist. Chaulmoogra oil was instilled into larynx one or two times weekly from October 23, 1922, until June 1, 1923, at which time she was discharged cured of her laryngitis as well as her pulmonary tuberculosis. Weight increased during these eight months from 140 to 185 pounds. There has been no recurrence.

While all of the Class A and B patients have recovered, two of the Class C patients died from pulmonary tuberculosis. Those who have recovered ran courses not materially different from those of Class B just reported. We shall, therefore, not devote time to them, but will summarize the two fatal cases because they showed continuous laryngeal improvement regardless of progressive involvement of the lungs.

Mrs. H. C. A., age 38, drove in an automobile from Michigan to Colorado in November, 1922, but did not consult a physician until February 28, 1923, and was seen in consultation by us the same day. History of having lived in Colorado for twenty years, moving to Michigan eight years previously. In March, 1920, had cough for two months, and in March, 1921, was given positive diagnosis of pulmonary tuberculosis after tubercle bacilli had been found in sputum. Daily temperature, 100 to 101 degrees for past seven months. Had some occasional "weakness of voice" seventeen years ago. Lost voice in September, 1922, and has not been able to speak since. Has constant pain in larynx, worse on inspiration, very severe on swallowing. Marked dyspnea. Examination of larynx showed epiglottis and arytenoids immensely infiltrated and edematous to the extent of prohibiting any view into anterior. Arytenoids and epiglottis were in apposition except for a narrow triangular slit, which hardly seemed large enough for any air to enter. Pulmonary diagnosis: Far advanced tuberculosis, bilateral (Class C). Prognosis: Hopeless. Laryngeal diagnosis: Tuberculous laryngitis, Class C. Treat-

ment: Chaulmoogra oil instillation into larynx twice weekly. Pain subsided after a few treatments of the oil and patient was comfortable as far as larynx was concerned until her death. Infiltration of arytenoids and epiglottis rapidly disappeared, later permitting a view of interior of larynx. Each cord and posterior wall was then found to be ulcerated, of the deep type, with considerable sloughing. These necrosed tissues were not benefited rapidly like the arytenoids and epiglottis, but slowly healed, and voice returned and remained clear and without huskiness, though weak, until her death, six months later, from tuberculous toxemia. The third day before she died the larynx was carefully examined, without discomfort or gagging on the patient's part, and was free from ulceration. Slight infiltration of epiglottis remained. The voice was clear. No autopsy was permitted, which, in my opinion, prevented positively proving that this larynx healed in spite of rapidly progressive fatal pulmonary tuberculosis.

Mr. F. H. P. came to Colorado on October 12, 1921, remaining here until his death from pulmonary hemorrhage, two years and six days later. Illness began six years previously with cough and general weakness. Weight down to 128 from normal of 140 pounds. Internists' diagnosis: Far advanced pulmonary tuberculosis, bilateral. Prognosis: Bad. Our examination as follows: History of hoarseness for past nine months. Much worse past three months, with intermittent aphonia, constant pain and difficult swallowing. Inspection of larynx as follows: Arytenoids, club shaped with great induration; posterior laryngeal wall, cushion of epiglottis, aryepiglottic folds, interarytenoid space, ventricular bands and vocal cords, all greatly infiltrated. The vocal cords were also roughened and "mouse nibbled" along free border by extensive ulceration. Diagnosis: Laryngeal tuberculosis, Class C. Patient was given routine treatment, including silence but not chaulmoogra oil, until May 9, 1922, seven months, during which time there was no general or laryngeal improvement, and the ulceration had extended to the right side of an enormously infiltrated epiglottis. View into interior of larynx at this time (May 9) almost impossible on account of induration and swelling of arytenoids and epiglottis. Weight ten pounds less—i. e., 118 pounds. Says pain is constantly severe, and

swallowing and sleep almost impossible. Cautery of epiglottis or removal advised and refused. Chaulmoogra oil treatments begun, two or three times weekly. Third treatment, patient says he is much better and eating. Fourth treatment, shows gain in weight of four pounds. Fifth treatment, larynx shows much less swelling and ulcers much cleaner. Sixth treatment, says he feels fine, eats and sleeps perfectly. Eighth treatment, weight now 128 pounds—i. e., ten pounds' gain. Tenth treatment, patient has decided, against all orders, to remain out of bed all the time, is driving automobile and has lost ten pounds. Feels badly. Twelfth treatment, again feeling fine; gained two pounds. Inspection shows larynx free from ulceration, only slight infiltration remaining; no aphonia or dysphagia. The larynx remained in this condition, with slight fluctuation, during the following three months—the last three months of his life—notwithstanding the fact that his pulmonary and general condition progressively grew worse. In fairness to his internist, it might be stated that during the last several months of this man's life he decided (like an occasional far advanced tuberculous person will) to have a "good time," regardless of consequences, and hence disregarded all advice. Death from pulmonary hemorrhage was not unexpected. The point of interest, however, was the improvement in laryngeal condition with simultaneous progress of pulmonary disease.[52]

The greatest factors, when considering causes for cure or improvement in the cases cited, are evidently rest, chaulmoogra oil and increased general resistance. Rest as a cure for early tuberculosis in any tissue cannot be discredited. Such would be contrary to the belief of many observers. In the last case cited, however, chaulmoogra oil seems, by exclusion, to have been the only factor contributory to laryngeal improvement.

Carmody,[50] too, has seen the larynx improve with the pulmonary condition progressing, as evidenced by his saying "at times the larynx will improve in the face of failure of the general condition."

In that some of the favorable reports of other writers quoted above were written almost two years ago, the writer has thought it wise to have the more recent ideas of those observers and quotes the following personal communications.

Dr. Robert M. Lukens, of Philadelphia, says on November 10, 1922:

"I am still enthusiastic about chaulmoogra oil and have learned considerably more about it. The chief indications for its use are dysphagia, ulcerations and tuberculous excrescences. Infiltrations with unbroken mucosa also benefit, but to a less degree. I have found the fresh oil, which I obtain direct from Parke, Davis & Co., is vastly better in its action than specimens I purchased from local druggists. Checking up these specimens through Parke-Davis' representative, I found that they were a year or more old; this probably accounts for the disappointing results obtained by some men. Use fresh oil and do not heat it, as heating seems to break it up and spoil it to some extent. If it is solid, due to cold, place it in a warm place and allow it to liquefy slowly, and I am sure you will be gratified by the results of its use. While some men have been disappointed with the oil, a larger number have reported results similar to my own."

Dr. Lukens again writes a year later (October 12, 1923), as follows:

"I consider chaulmoogra oil as the best routine medicinal agent in my clinic. Of course, in certain conditions I employ surgery and the cautery, but only in specially selected cases, as I consider surgery of the tuberculous larynx a risky business unless there is some very urgent need for it."

Dr. Samuel Swezey, of the National Jewish Hospital for Consumptives, on September 24, 1923, writes as follows:

"We have had considerable experience with chaulmoogra oil in the treatment of laryngeal tuberculosis. This work was stimulated by the reports of other clinicians in medical literature. The results with this treatment in our hands were rather uncertain. It seemed to have no effect upon the progress of the laryngeal conditions, and the only definite result that we noticed was the relief of pain in cases of dysphagia. Work along this line was also started in our Research Department by Drs. H. J. Corper and Harold Freed, who published their results in the Journal of the American Medical Association, November 18, 1922, page 1739. In view of the fact that the chaulmoogra oil treatment did not prove to be of special ben-

efit to our laryngeal cases clinically, and also in view of the findings of our Research Department, the treatment was discontinued."

Dr. I. D. Bronfin, superintendent of the Jewish Consumptives' Relief Society, of Sanatorium, Colo., under date of September 29, 1923, says:

"Our experience with chaulmoogra oil has been chiefly in connection with laryngeal tuberculosis. We used both the ethel esters of the oil, as well as the crude drug in varying strength, from 5 to 50 per cent, and in some cases we used also the pure oil. We use either the laryngeal applicator or the tracheal syringe. Our results with 50 cases for a period of nine months were rather discouraging. At present the result can be said to be negative. We failed to corroborate the findings of other authors, that chaulmoogra oil was capable of relieving pain and dysphagia. A complete report of this work will soon be published, at which time I shall be only too glad to send you a reprint. We are now planning to use this drug for its general systemic effect; but before doing that we will carry on this work with animals to make certain that no ill effects can result from its use."

Dr. James E. Lebensohn,[49] of Chicago, under date of November 11, 1922, says the following:

"We do not believe that chaulmoogra oil is a definite cure for all varieties and stages of the laryngeal tuberculosis, but it has a definite value in the milder forms of laryngeal disease. The use of the oil in certain types of laryngeal affections seems to be altogether without value. For example: In epiglottic infiltration, where the dysphagia is often extreme, we have found that relief of a permanent nature only follows in the surgical procedure of removing the infiltrated epiglottis. In this case orthoform will give some temporary relief, but we are quite enthusiastic about the value of the surgical procedure suggested.

"We have noted, as you probably have also, that certain writers believe that chaulmoogra oil produces a deleterious effect in the general management of tuberculosis, causing disquieting febrile reaction. The observation of the medical men in the tuberculous wards and our own analysis of the charts

do not support this contention. We have treated to date about 150 cases of laryngeal tuberculosis, and in none do we believe untoward general effects have resulted.

"We have used for the past two months, instead of the chaulmoogra oil, a preparation of the ethyl esters, known as chaulmestrol (New and Unofficial Remedies). We find that this is somewhat more pleasant than the chaulmoogra oil and produces less of a cough reaction. We have used it in 10 and 15 per cent solution in liquid petroleum. It, however, does not seem to be as effective as the chaulmoogra oil itself.

"Since the introduction of chaulmoogra oil as a routine use in our cases of laryngeal tuberculosis, it is only rarely that we have had to resort to the ameliorating remedies, as cocain, orthoform, anesthesin. We believe that the use of the oil is much more justified and probably more effective than the cautery methods used, such as lactic acid. We have found that when the affection of the larynx is in the lower third, producing aphonia, good results are obtained also by the judicious use of the oil. When the oil is used daily the cords are kept in a state of continual irritation, and the voice does not improve, but if we use the oil in weekly or biweekly treatments the voice very often improves and sometimes remarkably so.

"We had anticipated writing a new article on the use of chaulmoogra oil and chaulmestrol, with a further analysis of their effects and action. However, we do not believe that the oil occupies a sufficient and distinct place to give it overmuch publicity."

A year later (October 12, 1923) Dr. Lebensohn says:

"My further experience with chaulmoogra oil only more convinces me that it has a definite analgesic action on the larynx."

CONCLUSIONS.

1. Rapid advancement has been hindered in the treatment of laryngeal tuberculosis because of: (a) Lack of early diagnosis; (b) Indifference on the part of the physician, because of his ignorance as to the curability of the disease and because of his fear of being contaminated by the patient.

2. Voice rest, coexisting with the administration of nearly every remedy, and after enforced by Nature, may be a greater factor in the cure than the remedy itself.

3. Chaulmoogra oil, although condemned by some, seems to be the most valuable recent addition to a long list of remedies, having at least brought relief to many advanced sufferers and possibly cures to many earlier ones. In our own cases (though limited in number), it has seemed to be more efficacious than any other nonsurgical remedy employed, both in early and advanced cases. Since many other therapeutic measures, however, have met with much early favor, but have later come into disrepute, conclusions should not, as yet, be ironclad. From the results thus far obtained, however, laryngologists in general would seem to be warranted in giving chaulmoogra oil a thorough trial.

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VI.

THE RELATIONSHIP OF THE TEETH TO PATHOLOGY OF THE MAXILLARY SINUS.

By Louis E. Brown, M. D.,

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When undertaking to consider a relationship between the teeth and sinuses, especially the maxillary sinus, it is natural that we should first think of the relationship between the practitioners of the healing art who are specially concerned with affections of those anatomic parts—that is, between the dentist—or perhaps I might better say the oral surgeon—and the rhinologist. If either of these men is to do his work intelligently, and carry it out with the greatest satisfaction to himself, he must have a working knowledge of the specialty of the other.

This does not mean that the rhinologist should usurp the function of the oral surgeon, or that the dentist should undertake to practice rhinology; as a matter of fact, it means exactly the opposite of this. In order to realize when a patient needs the attention of an expert dental surgeon, the "nose and throat man" must needs have a clear idea of the limits of his own field. And by the same token, the dentist who is thoroughly familiar with the anatomy and pathology of the structures adjacent to those with which he is particularly concerned will be the first to realize when a condition should pass from his control to that of one especially experienced in the care of these same related anatomic parts.

Of all the accessory sinuses the maxillary—or, as it is more commonly designated, antrum of Highmore, or simply "antrum"—is the one in which the dental surgeon must of necessity be primarily interested. It should hardly be a matter of surprise then, that not infrequently the dentist should feel it within his province to undertake treatment of abnormal conditions in this sinus, and that he should perhaps resent the insinuation of the rhinologist that he is exceeding his authority.

The anatomy of the maxillary sinus is of first importance to both dentist and rhinologist. The normal opening of the antrum of Highmore is high up in the nose, in the area occupied by the middle turbinate bone, at the junction of the superior and internal walls; and in order that the secretions accumulating in the sinus may be properly and continuously evacuated, natural syphonage is essential. The antral opening is very minute, so when an inflammation takes place in the nose or the mucosa swells from any other cause, it can be readily appreciated how quickly this entrance may be occluded and drainage completely cut off.

The actual shape of the antrum, as well as its location in relation to the jaw, varies to a certain extent, even in normal individuals. Except at its lower internal portion, which is the outer wall of the inferior meatus of the nose, the antral walls are very thick. This lower portion is thin enough so that it is possible for the rhinologist to puncture it with a trocar when it becomes necessary to inspect the cavity. In some individuals the floor of the antrum may be on a level with that of the nose, but in the majority of patients it will be found lying considerably lower, so that the lower part of the cavity assumes the shape of a pocket, ready to act as a catch basin for those secretions which may be dammed back by some abnormal nasal condition. It is, of course, the antral floor which comes in close contact with the teeth; and so close is this relation that very often the roots of the teeth spread through the bony wall and penetrate the sinus.

The maxillary sinus is lined by an exceedingly fine mucosa, consisting of three layers: a ciliated epithelial, tunica propria and periosteal, and it is unusually susceptible to infection and subject to inflammatory reaction. At times inflammatory reaction within the sinus itself may be so great as to result in a thickening of the mucous membrane to the extent that actual polypoid formation may take place, without having an outside lesion to account for it.[9] The effect which abnormal conditions in the sinus may have upon the entire systemic condition is only just beginning to be appreciated, and but very little work has so far been done on the bacteriology of the accessory nasal cavities. Cary and McGinnis,[2] who have investigated quite widely along these lines, have recently re-

ported a case of bronchial asthma where marked improvement followed the clearing up of a double maxillary, ethmoid and frontal infection. Washings from the antrum showing streptococcus viridans in pure culture, before the beginning of treatment, and the author wishes to report a case of cholecystitis, accompanied by a marked jaundice and severe gastric symptoms which had existed for several months, the case having previously been diagnosed as an acute gastritis and treated as such. This case cleared up very promptly, upon finding and instituting the proper treatment of the primary source of infection, which existed in the right maxillary sinus.

Because of its anatomic position the antrum forms a ready reservoir for any discharge which may originate in the frontal or anterior ethmoid sinuses lying above it. "The normal opening of the antrum invites the pus from the frontals and anterior ethmoid cells to enter and find a most suitable field for propagation. This large reservoir, with its overflow at the top, is certainly an ideal incubator. . . . An infectious discharge from any of the sinuses may infect the throat and thus be the means of producing serious secondary symptoms, or the pus may be swallowed with resultant gastrointestinal infection. The antrum, however, when infected, is the most fruitful source of systemic infection by direct absorption into the circulation."[4]

Looking at this from another angle, Hays of New York[9] contends that though it is possible for chronic antral conditions to be associated with chronic conditions of the other nasal sinuses, it is far more likely that the majority of chronic cases of antral diseases arise from some necrosis of the jaw or teeth. Sinus thrombosis, often due to suppuration in the middle ear, is also a frequent sequel to other pathologic conditions about the head or face, among which dental caries has been recorded as an etiologic factor.[3] It is a "fashion" just at present to refer a large proportion of human ills to dental infection, but it is well to remember that the primary trouble may not reside in the teeth, and only a careful consideration of all the factors will enable the diagnostician to reach a correct conclusion.

That dental caries is one of the products—I might almost say the drawbacks—of civilization is now widely recognized.

Siffre,[11] who examined a large number of neolithic skulls found in a mound near Seine-et-Oise, observed seven superior maxillæ showing perforation of the maxillary sinus, very evidently the result of a dental lesion. These perforations had apparently come about through trauma—that is, fracture of the teeth, which permitted the entrance of infection to the pulp cavity and the root, with consequent empyema. Caries seemed to follow fracture of the teeth in the well understood classic order: pulpitis, infection of the pulp carried to the alvodental articulation, apical cyst or granuloma, abscess, periodental necrosis and fistula. But it was only by trauma or by use that primitive man lost his dental equipment, the teeth showing little or no evidence of caries as we are familiar with it today.

The manner in which modern civilization affects the structure of the jaw, which in turn reacts not only upon the teeth themselves, but also upon all adjacent bony structures, has been recently well described by Dr. W. H. Haskin of New York.[8] Though the prevalent malformations of the jaw may be due in considerable measure to congenital influences, more frequently it is lack of stimulation from mastication which retards proper development. This writer believes that the greatest of all causes in producing malocclusions with the resulting asymmetric developments of the face, causing nasal obstructions (a frequent source of closure of the antral opening) and orbital troubles, is the lack of full muscular efforts in feeding. This begins in infancy and continues practically throughout life, especially in this country. The bottle fed infant spends about 64 minutes a day in sucking his ration of milk from a rubber nipple, while the baby who gets his nourishment in the natural manner must exert his sucking powers for 20 minutes at a feeding, or about 160 minutes in 24 hours. The Indians and those foreign immigrants whose previous environment did not give them access to refined flour and well cooked cereal foods, and among whom infants are universally breast fed, have uniformly excellent teeth; yet one generation reared under the "softer" conditions of our civilization will show a marked dental deterioration, simply from lack of sufficient exercise of the jaws which should induce stimulation of the dental germs, and thus aid in developing strong, healthy teeth, with firm alveolar processes to hold them in place. Where

forward and downward growth has been retarded there will be a marked vertical bow in the nasal septum, "which is probably explained by recognizing that the vomer belongs to the cranial bones and grows to correspond in size with them, only to find that the maxillæ have not grown sufficiently and there is consequently not room enough for the vomer, as a result of which it is forced to bend." And nasal obstruction, as we have already seen, is one of the frequent causes of antral disease.

Congenital malformations of the teeth and jaws may affect the antra in still other ways. Stumpf,[13] in discussing a case where, at the age of 39, a patient developed a cyst of the jaw which a roentgenogram proved to be due to a canine tooth under the floor of the right nostril, calls attention to the frequency with which premolars and molars may be misplaced into the antrum; and often—as in the case cited—give no symptoms until adult life is well advanced. Impaction of the teeth is most frequently due to the same causal factors which we discussed above, those arising from improper exercise of the jaws. When such teeth have wandered into the antrum they may be the cause of obscure neuralgias or productive of empyema, even going on to fistula. Here roentgenography may be of great service in establishing a diagnosis.

Infection in the maxillary sinus—whatever its original cause may have been—is undoubtedly of far more common occurrence than is ordinarily recognized. The increasing employment of the X-rays in dental diagnosis has brought to light many an antral infection which had been previously unrecognized because it gave no frank objective symptoms.[7] In reporting 50 cases of infection of the maxillary antrum, Ayer and Buff[1] recently stated that "acute empyema of the antra is most common of all diseases of the sinuses," to which they appended the observation that a large number of such cases had—in their experience—their point of origin from the teeth, though only 25 per cent of antral infections are caused by carious teeth projecting into the antra. A recent writer[6] thinks that infection of the maxillary sinus is far more frequently of the ascending type—that is, due to dental conditions—than it is of the descending type, subsequent to infection which has drained down from the frontal and sphenoid sinuses or the

ethmoid cells. "It is very seldom," he remarks, "that we find a chronic maxillary sinusitis in a case where the dental line is unbroken. If the teeth are examined and a careful history regarding them obtained, it will be found in a great majority of cases that a first molar tooth, more frequently, and often the second molar tooth, or both, have been extracted. Many crowned or filled molar teeth whose pulp and nerve supply has been killed will also be noted."

J. Parsons Schaeffer of Philadelphia, noted anatomist of Jefferson Medical College, says that after the examination of hundreds of skulls he is convinced that the second molar tooth protrudes through the alveolar process into the antrum and is the cause of infection in the sinus more often than is generally supposed; and that before the use of the X-ray in dentistry the first molar tooth has always been the one to be condemned and extracted.

In the above remarks I have referred purposely to the molar teeth. But it must not be forgotten that any tooth from the central incisors back may be the cause of a maxillary sinus infection. This particularly applies to the lateral incisors, as an infection at the root of one of these teeth can quite readily, and frequently does, burrow its way through the cancellous bone above it into the antrum, a point well worth remembering when examining the teeth in search of the primary foci of infection.

Of course we have all quite agreed to the fact that the most frequent source of infection of the antrum arises in the nose. But as we are dealing here with the subject of the teeth as the primary foci of infection, the above statements and percentages pertain only to that source.

The diagnosis of these obscure cases of maxillary sinus infection naturally presents many difficulties and requires a very careful examination of the case. The routine followed by the author in attempting to make a diagnosis, which is somewhat similar to the one set forth by Spielberg, is as follows:[14] 1. The clinical history leads towards a suspicion of maxillary sinus involvement. 2. Palpation of the area over the antrum will often elicit pain. 3. Clinical findings within the nose, such as congestion and turgescence of the soft structures and the presence of pus. 4. If no secretion is present in the nose, the use of suction will often be found quite helpful. 5. By trans-

illumination of the antra you find the affected one to be hazy or cloudy, depending somewhat upon the extent of the involvement. 6. The X-ray picture will corroborate the above findings. (This X-ray picture in many cases should include the teeth as well as the antrum.) 7. Irrigation through the normal opening will usually determine whether there is pus present or not. 8. If impossible to irrigate through the normal opening, the antrum should be punctured and irrigated. The washings will as a rule definitely decide the question of involvement. 9. The author wishes to denounce the extraction of a sound, healthy tooth for the purpose of making a diagnosis, as this has long since been abandoned as obsolete.

The use of the nasopharyngoscope, as recommended by Skillern and others, which is inserted through a puncture in the outer nasal wall, will greatly assist in making a correct estimate of the existing conditions. The observations made by this inspection will be useful in checking up the X-ray findings which, when taken alone, are seldom absolutely reliable, and it also permits continuous study of the tissue changes in the antrum. Spielberg has found that when the X-ray plate shows clouding it almost invariably means that there is some pathologic process going on within the antrum; but whether it is due to acute congestion, atrophy with fibrous changes in the mucosa, polypoid degeneration, hypertrophied mucous membrane, a tumor, or fluid, can only be determined by an antroscopic examination of the sinus involved. The degree of clouding on the X-ray plate is no indication of the extent of the disease or involvement present, as an acute congestion of the mucosa will give a marked clouding, while with an extensive polypoid degeneration it may be only slight.

Some obscure lesions may, however, escape even this scrutiny. It has been pointed out by Sweet[15] that many obscure reflex symptoms should properly be attributed to sinus disease; for example, a right maxillary sinusitis may manifest itself by a left supraorbital pain, so that in cases of acute sinusitis the attendant should not be misled by the location of the pain, "but should seek by every method available to locate the offending sinus and to make sure that it is the only one, because several sinuses are frequently involved at the same time."

When the diagnosis has been established, either by the customary methods of transillumination and examination of washings or some of the more extraordinary measures necessity may force upon us, the question of treatment becomes of paramount importance. It is my personal opinion that in a large proportion of antral infections the teeth have been the exciting cause, but that the extraction of the offending tooth should not be regarded as a measure for producing an opening through the alveolar process for drainage and treatment. When the source of infection has been extirpated, it is, in the general run of cases, far better to handle the sinus infection intranasally, and, realizing that the conduct of the case is the concern of the rhinologist rather than the dentist, follow out the line of treatment ordinarily pursued in any sinus infection.

When we consider that nature intended to keep the maxillary sinus clear, not by drainage from its lowest point but by syphonage from a point very far up in the nose, the usual argument for breaking through the antral floor so as to empty it through the mouth—"at the lowest point"—will be realized as being wholly unsustained. In closing a lecture on the treatment of antral infections which Crane of Brooklyn[4] gave a few months ago, he stated that he wished "to unqualifiedly condemn the usual dental operation of extracting a tooth and cutting into the antrum through the alveolar process. A very fair proportion of antrum cases coming for operation have been submitted to this unsurgical procedure, with, of course, the failure to effect a good surgical result." A similar view is held by Crebbin of New Orleans,[5] who says that "the extraction of a tooth for drainage of the maxillary sinus cannot be too severely condemned, unless a diseased tooth or necrosis of the alveolar process is responsible for the discharge. This should be the only cause for resorting to such an unscientific procedure."

Patients have come to the author, who have had a tooth extracted, and so much curettage has been done about the root opening, that a large hole was broken through the alveolar process into the maxillary sinus. I can recall two cases in particular, where it required a year or more to close up the opening thus made. The difficulty of eradicating such a fistula is very great. It has been recently noted by Maybaum[10] that though an alveolar sinus of small caliber responds readily to

treatment with cauterizing agents, when the antrum infection has been eliminated, the obliteration of large openings is a much more complicated and tedious matter; and, in his opinion, "can only be accomplished by means of some plastic operation, together with an intranasal opening or a radical antrum operation with a nasal counter opening to provide for thorough drainage through the nose."

By closure of the alveolar opening—assuming that one exists either through ill advised dentistry or the invasion of the sinus by one of the molars, or from some other accident—we cut off a fertile source of infection from below; and by performing a proper intranasal operation we obtain and maintain, permanently if necessary, as is needed in chronic cases, an opening into the antrum whereby we are assured ease of medication and free aeration, which, to my mind, is of the utmost importance in all infections of this sinus, and is impossible to obtain when intervention is undertaken through the oral cavity. While in chronic cases with polypoid degeneration of the lining membrane, a tumefaction of some type, a foreign body or an extremely virulent infection in which a radical operation is done through the canine fossa, it is very necessary to have the counter opening into the nose, rather than through the alveolar process, in order to secure drainage, medication and free aeration over quite a period of time.

I have purposely refrained from suggesting any specific routine method of surgical procedure, but have strongly advocated a few things that are essential and of the utmost importance in any operation performed on the maxillary sinus.

To summarize the above I would say:

First, that there should be a more earnest and hearty cooperation between the oral surgeon and the rhinologist in order to obtain earlier and better results in maxillary sinus infections.

Second, more care should be exercised in attempting to find the primary foci of infection and making a correct diagnosis.

Third, that as normal drainage of the antrum is obtained by the process of syphonage, hence, in most cases of maxillary sinus infection, the treatment of it rightfully belongs to the rhinologist.

Fourth, that the danger of reinfection through the tooth socket is far greater than through the nose, hence when an antrum infection is treated by this route rather than the latter, we encounter a decided handicap to early recovery.

Fifth, and most important, that only by the intranasal route can ease of medication and free aeration be obtained, these being two of the most essential points in the early and permanent relief of maxillary sinus infection.

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VII.

VESTIBULAR ACCOMMODATION AND A DESCRIPTION OF THE QUANTITATIVE CALORIC REACTION OF BRUNINGS AND KOBRAK.

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It is not surprising that the average otologist has but little practical knowledge of the testing of the static labyrinth, for though the subject is an old one, the maze of literature and of findings leaves but one impression—confusion. This does not alter the fact that we are in frequent need of a good and simple test to determine the function of the labyrinth. Kerrison, in the last edition of his Diseases of the Ear, has set down what is the most lucid and practical exposition of the tests and the reasons for the reactions. He emphasizes a fact which in no other writings has been given sufficient force—this is, the early accommodation of the system to disturbance of orientation. A digression can well be made to quote Kerrison before taking up the description of the test that has not yet appeared in our literature. An understanding of accommodation will frequently explain the findings in this test, as it will in other vestibular tests.

"That the vestibular organs cannot be regarded as the essential organs of orientation is shown by the fact that after removal of one or both labyrinths the individual regains in time his equilibrium. There must be, therefore, other contributory factors, and these are found in the muscular and arthrodial senses, the tactile sense and the sense of sight. . . . When sight is lost the arthrodial and muscular impressions become more sensitive and accurate. When one vestibular apparatus is destroyed, the opposite labyrinth must become accustomed to unopposed, unilateral activity, and the muscular, arthrodial and tactile impressions of space and position must be immensely increased. Until this process of involuntary edu-

cation is well advanced, it is not surprising that the individual experiences occasional vertigo and ataxia, though he does not exhibit nystagmus and may show no constant tendency to fall in any given direction."

As these cases of labyrinth trouble come to us they may be put into two classes: (1) the acute, where no accommodation has had time to develop, and (2) chronic or long standing, where accommodation may be partial or complete. Spontaneous nystagmus has usually disappeared from the case if the onset of the disturbance has occurred one or two weeks previously, and it is therefore to be dwelt upon as merely an evidence of acuity, not of grade of reaction. The cause of the trouble may continue to be as active as in the incipiency, and our efforts at determining degree of involvement must depend upon the degree of reaction which our tests produce, looked at in the light of the duration of the condition.

Caloric reaction will be found excessive in acute or subacute stages of congestive, toxic or suppurative labyrinthitis; in old, accommodated conditions, and in nearly all types of chronic syphilitic involvement of the eighth nerve, the reactions will be subnormal or totally lost. Many of the cases that have been of long standing but that have not progressed to total loss of vestibular branch function will show temporary periods of increased irritability. At this time the patient will complain of transitory vertigo and unsteadiness, rarely nausea. The condition is followed usually by a further retrogression in vestibular responsiveness. Kerrison has described one type that, after accommodation has taken place, will show a vertigo on assuming unfamiliar positions, such as leaning backwards or looking at the ceiling. But the aggravation that I refer to above is not of this type; it appears without any unusual provocation and is due to the inherent pathology behind the condition.

With the fact of early accommodation before us, it is obvious that the better conclusions can be drawn from earlier tests. The next problem is that of the selection of the best type of stimulus to use to elicit in a measurable manner the degree of vestibular irritation or destruction. Of the three cardinal manifestations of vestibular disease, nystagmus, vertigo and falling, the first is alone available for accurate testing pur-

poses. Up to this time it has been universal to use Barany's method of mass stimulation both for the caloric and turning tests. But a change is taking place, and many European authorities are advocating the use of minimal stimuli in order to determine the threshold of nystagmus. Gradenigo[6] in Italy has been using a single turning for the turning test, and the caloric threshold reaction test has been fostered by Beck, Brünings, Kobrak, Grahe, Krproff, Michaelson, Quix and Wojatschek. The test bears Brünings' name in Kobrak and Jansen's book.[17]

It is apparent that these men believe that the end point of the passing of nystagmus is indefinite on account of gradual disappearance. They are not altogether satisfied with Barany's mass stimulation and the timing of the duration of the nystagmus, and they believe that much more can be learned from he use of minimum stimulus and from the observation of the time elapsed before reaction begins.

Translation of Kobrak's description of the test (the parentheses are those of the author):

"Brünings has succeeded in determining the quantitative caloric reaction and has described an optimum position of the head for the stimulation of the horizontal canal. The latter is as follows: Bending the head backward about sixty degrees, it is slightly inclined toward the shoulder of the douched side. In this best position (best, because the horizontal canal is most nearly vertical) the circulation of the lymph is possible. (There can be no circulation in a horizontal system by the displacement of water of different temperature. Brünings lays stress upon selecting an optimum position of the horizontal canal because this canal by its anatomic position is nearest the source of the temperature change, that is, most externally placed.)

"Then having put the head in this position, Brünings uses a special irrigating apparatus and allows water at 27° C. (82° F.) to flow slowly into the ear until the first twitchings of the eyes start. This happens in most cases after 65 to 70 c. c. of water has been used. This amount designates the stimulation quantum. Now, the amount of water required depends not only upon the irritability of the organ of equilibrium, but also upon the 'cold carrying disposition' (the temperature transmission) of the middle ear. One has then to look for a way to measure the stimulation and to register the duration

of the nystagmus. The duration of the nystagmus is indeed apparent, not so much by the irritability of the end organs, which interests us before all, but it depends upon central ("kern") factors in such a manner that the central stimulation works automatically and in a certain degree independently of the spreading irritation.

"A reliable quantitative measurement of the excitability of the vestibular apparatus seems to be possible through a modification of the method.

"Everyone who has made many caloric experiments according to the method used formerly, with large quantities of water, 70 c. c. and more, must see that the weak initial nystagmus, often hard to perceive, will little by little become stronger after washing has started, also after further factors of irritation have been in evidence. This makes us involuntarily wonder whether these initial twitchings of the eyes show merely the beginning of the reaction or whether the reaction had already started during the washing. This caused me to adopt the following method:

"Basic temperature of the water is maintained at 70 degrees. Begin douching with 5 cc. Then wait a few seconds to see if any weak nystagmus appears. Should there be no reaction wait at least one minute in order to allow the small currents of lymph or other occurrences of reaction to come to rest again.

"Then try with about 10 cc. to obtain the reaction ("schwellenwertsreaktion"). If with 10 cc. no reaction has started, a pause of at least five minutes is made in order to let the lymph circulation and the irritation of the vestibule cease. Then try with 20 cc. Should a reaction occur, try between 10 and 20 cc. —15 cc. If again there is a reaction, try between 10 and 15 cc. —13 cc. If no reaction, try between 15 and 20 cc.—18 cc. (In such manner the exact number of cc. necessary to produce a reaction is found.)

"I was astonished when by means of the quantitative measurement I found the threshold to be so sharply defined that very small differences in quantity of water brought it out. (An addition of two and a half cc. of water to that previously used is often sufficient to pass from the submminimal to the minimal stimulus in the normal labyrinth.)

"The method is simple for quantitative measurement; an ear syringe is always in the equipment of every physician. With the 'einschrankungen' to be expected, it is accurate enough and has the undeniable advantage that with this very small initial stimulation of the vestibule, generally no sensations of dizziness manifest themselves. We ascertain by this method of threshold determination, first, the minimum water (at a given temperature) that is necessary to start a reaction; second, we determine the time from the beginning of the douching until the beginning of the nystagmus, which (nystagmus) may not be physiologically strong enough to be taken as the duration of the reaction but may perhaps be accepted as the extended time of the reaction."

Roughly, then, it can be seen from the above translation that (1) Brünings has found an optimum position in which to place the head during douching. (2) Kobrak has modified Brünings' quantitative, measured, but massive douching to a quantitative, measured, but minimal douching—using just sufficient water to produce a reaction. Both use 27° C. (82° F.). In the cases here reported I have adopted the position of Brünings and the technic of Kobrak, but instead of water at 27° C., I use 10° C. (50° F.) or 15° C. (60° F.). This is done because a greater difference in temperature below the body temperature (36.9° C.) gives a stronger stimulation per cc. of water used; this should make the threshold as to quantity of water used sharper, the reaction more distinct. It is for like reason that hot water douching is not used. Water at 115° F. (45° C.) begins to cause pain on douching; and there is not the opportunity to use great difference in temperature above the body normal as there is below the normal. The accompanying table graphically depicts this, and will be found useful in transposing temperatures from one scale to the other.

Certain clinical observations are important to note.

1. It is not necessary to use Brünings' position to get caloric reactions, but it is advisable. It is essential to maintain a constancy in technic. Changing the position of the head (after douching) alters or stops the nystagmus; not so with the turning tests.

2. Careful attention should be given to the recovery period after subminimal stimulation, and consecutive douchings should

not be carried on without full rest periods between them. With the repetition of subminimal stimuli, without rest periods, it is possible to demonstrate that the labyrinth reacts physiologically as do other responsive human tissues. Subminimal stimuli accumulate until they are equivalent to a minimal stimulus.

3. The rapidity of the douching seems to make little difference in the findings; the small quantities used allow one to complete the douching in five or ten seconds at most, an almost negligible factor.

4. The temperature of the syringe should be that of the water, and this can be maintained by leaving the syringe standing in a beaker of the water.

5. Focusing on a near point diminishes or checks the nystagmus. The patient should relax the eyes and look casually, not intently, at a distant object. The approach to the threshold may be surmised by the appearance of a nystagmus when the patient is made to look to the left when the right ear is douched; to the right when the left ear is douched. That is, the patient is directed to look in the direction of the recovery or quick component.

6. Threshold stimuli in their effect upon normal labyrinths rarely produce any subjective symptoms.

7. The extent of the excursion of the nystagmus seems to bear no relation to the amounts of the minimal stimulus required. Given an impaired labyrinth with a slow reaction, requiring a minimal stimulus of 60 cc. of water at 10° C., the nystagmus set up may be very slight or marked. This seems also true to a less degree of the hypersensitive labyrinth. However, here nystagmus is more apt to be of wide excursion. There is no set rule of behavior observable at the threshold.

8. In diseased labyrinths, whether hypo- or hypersensitive, the degree of accommodation that has taken place is of utmost importance in judging the findings.

Complete accommodation to vestibular loss may occur very rapidly (one to two weeks) or it may appear more slowly (one month). This does not depend upon the causative pathology to the extent that might be supposed. As example: two cases of fracture of the petrous pyramid. One had vertigenous spells for two days; another had moderate symptoms for two months. An acute suppuration of the middle ear may affect (without

direct extension) the labyrinth, and may cause a permanent, demonstrable loss of function—but in such a case there may appear no vertigo during the active course of the disease. In another such case, vertigo may be mild or severe. A luetic labyrinthine function loss may be so gradual as to produce no vertigo. Accommodation keeps pace with the slow destruction. The terminal findings may be complete loss and complete accommodation.

9. If the labyrinthine pathology is on one side only, the healthy labyrinth on the opposite side usually partakes in the accommodation, and in time may become irresponsive to caloric and to turning tests. The phenomenon, from the point of view of the normal side, must be one of negation of central perception, such as occurs with the visual stimuli that come to the weak eye in squint cases. With the vestibular reactions this fact can be repeatedly verified, yet there is but occasional reference to it in the literature, foreign or domestic.

10. Many cases of slowly progressive labyrinthine loss of function show acute exacerbation of symptoms—vertigo. During these times the picture changes from that of a dead or nearly dead labyrinth to a more or less active labyrinth. Yet in most cases, where the loss has been great, the exacerbations rarely show test reactions more marked than normal.

11. There are many foreign observers that report upon conditions that affect the conduction of heat and cold to the labyrinth. It is obvious that cerumen, furunculosis and a narrowed external auditory canal will affect the test conditions.

12. There is also much foreign literature on the question as to whether or not the douching affects the end organ or the nerve as well. This question cannot have the importance when we are testing with a minimal amount of water as when mass douching is used.

13. Brünings believes that the duration of the nystagmus depends not so much upon the irritability of the end organ as upon central factors. It is well, however, to note the duration, at least for the sake of collecting information on the subject.

Though in the cases about to be cited there are some general conclusions made, there is no attempt to particularize upon the pathology responsible for the findings. The purpose of the paper has been rather to describe what appears to be a valuable

test than to treat of causes. Interpretations should require lengthy consideration and discussion. Results are here put down just as they appeared. The cases have been selected to illustrate accommodation.

It is interesting to note, however, that the comparative findings between the caloric reaction and the turning tests tend to agree. In reading the valuable article by Kopetzky and Almour (Laryngoscope, April, 1924), one is led to believe that there should be found differences in the results of these two tests. There is sufficient evidence to show that the phenomena arising from each, "though alike in their appearance, are totally unlike as regards their production and true origin." The experiments of Borries are responsible for the present day distinction between the two tests. Borries found that on destroying the semicircular canals in pigeons he could still get caloric reactions but not turning reactions; he concluded that the phenomena of the caloric test arose from the otolith organs. The discussion of Kopetzky's paper is very illuminating.

Case 1.—G. H. D., age 53; occupation, machinist; cc. deafness. F. H.: Brother deaf after scarlet fever; no other deafness in family. P. M. H.: Pneumonia 25 years ago; general health has been good, but has been deaf "more or less" since childhood; the last 10 years it has been getting worse. There has always been some ringing in the ears. The ears have never discharged. He has always worked as a machinist, in the presence of much noise. In addition to his deafness he has for years been troubled with headaches; they come on frequently and are localized to the right forehead, right eye and right ear, "always settles in the right eye and right ear." He is very subject to head catarrh. There is evidence of circulatory insufficiency in dyspnea on exertion and in coldness of the extremities; occasional cramps in legs. He has been under a good deal of nervous strain in the last few years. Denies venereal history.

Present condition: As outlined above, deafness (marked), tinnitus, circulatory insufficiency, and evidence of nerve strain.

Examination: After impacted cerumen was removed from both sides, the drums appear thick, lusterless, opaque, immobile and not retracted. The tubes politzerize easily; there is a marked deflection of the nasal septum obstructing the right

nostril. The rest of the tract shows no pathology. He does not lip read and appears very slow at perception.

Hearing: Loud spoken voice heard at each ear. Galton whistle, right 8.8; left 6.7. Weber unreferred. Forks, AC and BC both reduced, but the reduction of the BC is in greater proportion. Audiometer, considerable loss (symmetric) for pitches up to 512, great loss above this. Argyll Robertson pupil positive. Slight ankle clonus, Romberg negative, exaggeration of all tendon reflexes. Blood pressure, 160—120; slight roughness to the first heart sound, heard best over the ensiform.

Caloric reaction:

Right ear	30 cc.	15° C. no reaction, no dizziness.
	60 cc.	15° C. no reaction, no dizziness.
	90 cc.	15° C. no reaction, no dizziness.
	60 cc.	48.75° C. no reaction, no dizziness.
	90 cc.	48.75° C. no reaction, no dizziness.
Left ear	15 cc.	15° C. no reaction, no dizziness.
	30 cc.	15° C. no reaction, no dizziness.
	60 cc.	15° C. no reaction, no dizziness.
	90 cc.	15° C. no reaction, no dizziness.
	60 cc.	48° C. no reaction, no dizziness.
	90 cc.	48° C. no reaction, no dizziness.

There is no nystagmus or dizziness after douching with cold or hot water, nor is it brought on after douching by looking at the ceiling or floor. The static labyrinth is absolutely unresponsive to the caloric test.

Turning tests: There is no response to the turning tests, no matter how they are applied as to the number of turnings or as to the position of the head. There are no subjective symptoms produced.

Past pointing: There is no past pointing after either the caloric or turning tests.

Conclusion: The static labyrinth is badly, if not completely destroyed. The auditory branch has suffered severely in the disease. The onset and course of the condition have been so slow that no subjective symptoms were produced. Accommodation likewise has taken place gradually but completely.

Case 2 (Dr. Gleason's Clinic).—A healthy young colored man was struck on the head with a beer bottle and was brought

into the hospital bleeding from the left ear and unconscious. X-ray picture showed what the radiologist said was a fracture through the left petrous pyramid. On regaining consciousness there was marked vertigo, nystagmus, tinnitus and deafness in the left ear. Unfortunately for this report, no careful examination or description of the ear findings or the nystagmus was made. When seen one month after his "accident" he complained merely of the deafness of his left ear. He said he had continuous dizziness for five or six days, so severe that he could not move in bed without getting sick. This gradually wore off; the attacks became less severe, became periodic, and finally would only appear when he turned or moved his head quickly. Examination after accommodation had taken place showed total nerve deafness on the left, absolutely no reaction to the turning tests, no matter how varied, and absolutely no caloric reactions from either ear. The good ear showed only the slight loss of the borrowed bone conduction.

Conclusion: The left labyrinth is destroyed completely; accommodation has taken place completely and rapidly. In this compensation for a unilateral destruction the possibility of stimulating the sound labyrinth by the turning test or by the caloric test disappears.

Case 3.—John S., age 39; spinner. A very alert and intelligent Italian, complains of dizziness, vertigenous attacks that come on irregularly without relation to any other symptoms or conditions. His general health is and has been excellent; he has always been well. The duration has been only a few months. There is no tinnitus, no deafness, and no spontaneous nystagmus. Venereal history is denied. Reflexes are normal, no Romberg; eye reactions are normal. Wassermann (?). There is no visible pathology in the nose, throat or ear, and general examination is roughly negative. Hearing tests show excellent hearing.

Caloric reactions:

Right ear 10 cc. 12° C. no reaction, no dizziness.
30 cc. 12° C. no reaction, no dizziness.
60 cc. 12° C. in 1 minute a rotatory nystagmus, quick component to the opposite side, lasting 30 seconds, no dizziness, recurrence 2 seconds after subsiding on looking to left.

Left ear 10 cc. 15° C. no reaction, no dizziness.
30 cc. 15° C. no reaction, no dizziness.
90 cc. 15° C. immediate reaction lasting 2 minutes, no dizziness.

Turning tests: No reaction with the usual technic.

Conclusions.—The cause and the true pathology of the case are obscure. The turning test response is nil, but the caloric test shows still some vestibular function. The caloric reaction is practically identical on both sides; hearing is unimpaired. Accommodation has almost completely taken place, yet the patient complains of attacks of vertigo, at which time there will probably appear a more actively responsive labyrinth. Many cases show this reawakening of an apparently nearly dead labyrinth. As the hearing in this case is bilaterally good, as there is no history of nor visible pathology in the ears, and as the vestibular loss is practically symmetric, the suggestion is had that the cause is some central pathology of the vestibular routes or tracts.

Case 4.—Samuel G., age 44, tailor, alert and intelligent, complains of tinnitus in the right ear. The condition has been of long standing and appeared gradually "without cause." Previous medical history and family history are negative. General examination shows no noteworthy pathology. The reflexes are normal, and Wassermann and provocative Wassermann were both negative. Ear examination is negative, except that both tubes are with difficulty politzerized. Hearing tests show a moderate catarrhal deafness on the left side, while on the right the hearing is equally poor up to 2048 d. v., where hearing suddenly drops out.

Turning tests elicit absolutely no reaction, either subjective or objective.

Caloric test: No reactions and no symptoms to 10 cc., 20 cc., 30 cc., 60 cc., 90 cc., in either ear (cold water at 50° Fahr., 10° C.). Turning tests produce no reactions and no symptoms.

Conclusions.—The unilateral loss of hearing indicates that the pathologic condition is but one sided, in spite of the bilateral loss of the vestibular reactions to the caloric and turning tests. The deafness is a unilateral nerve affection of the right side, cause unknown, though syphilis is suspected in spite of

the negative Wassermanns. (Hearing is improved and tinnitus somewhat relieved by mixed treatment.) The left vestibular apparatus shows no reactions, for the reason that compensation has completely taken place for the disease on the right side. Not only is there no longer an overbalance of an active side over an inactive side, with no longer the restraint of bilaterally coordinated processes, but now the uninvolved side is incapable of being aroused to produce a vertigo. This does not presuppose any pathology on the good side; it rather speaks for a central adjustment that practically puts the whole static-labyrinth in the discard.

Case 5.—Emily H., age 14, had been slightly deaf for some years. Until seven months ago she could hear the spoken voice without much error. She then developed a rapid increase in the deafness, until now she hears practically nothing. Wassermann is "moderately positive"; knee jerks and Achilles reflex are normal; slight ataxia, tends to fall to the right; Romberg positive. Pupils, which are unequal, show the Argyll Robertson reaction; eye movements are normal.

Hearing tests: Patient says she hears the Weber on the right, but she is uncertain. (A large 512 fork, with heavy vibrations, was used.) No hearing by air conduction in either ear to the fork; B. C. on the right gone, reported as very slight on the left mastoid (probably tactile). With the Western Electric audiometer No. 1, there is about 95 per cent loss in the right ear and 90 per cent loss in the left.

Caloric reaction (90 cc.), three ounces of cold water, at 50° F. (10° C.) produces no reaction or subjective symptoms on either side.

Conclusions.—Bilateral total involvement of the eighth nerve. In the acute exacerbation of the disease during the past seven months the vestibular involvement was not sufficiently strong at any time to produce vertigo. Accommodation is not yet complete, for there is still a tendency to fall to the right. Hearing tests show that this side is slightly more involved. The visual, tactile and arthrodial senses have not yet completely supplanted the vestibular mechanism, and further, they are probably somewhat held up by a central nervous syphilis.

Case 6 (Dr. Stauffer's service).—John Solomon, age 16, clerk. This robust, healthy lad came to the dispensary complaining of deafness in the left ear and tinnitus. As to his previous medical history, he had never suffered from any serious sickness or complaint other than incidental conditions; there was no history of an ear discharge. In August, 1924, he was knocked down by an auto truck and was taken to the Presbyterian Hospital, where the diagnosis of fracture of the base of the skull was made. The hospital notes at this time show that he was brought in unconscious; there was no bleeding from the ears but the nose bled freely. The X-ray report confirmed the diagnosis of a fracture through the petrous pyramid (left). He noticed deafness in the left ear when he recovered consciousness 24 hours after the accident. On the sixth or seventh day he vomited and was very dizzy. He was in bed seven days and was slightly dizzy for two days after he got up from bed. His hearing has never returned; left ear alone is deaf. One week ago (February 16, 1924) a tinnitus started; it was high pitched, "resembled the ringing of a bell," comes at any time of the day, does not keep him from sleeping nor does it awaken him. Family history is negative.

Examination: Is completely negative as to findings in the nose, throat and ears. The drums are in every respect normal. The only objective symptom is the deafness, the only subjective symptoms are the deafness and the tinnitus.

Hearing tests: There is absolute deafness for whispered and spoken voice at the left ear; the right ear hears normally. Brow to brow the voice sounds are carried to the right ear, yet the Weber with the fork is doubtfully referred to the right. Localization of sound is fair in all directions, even though the left ear does not hear.

Fork B488: Right ear, A. C., 60″ (n/—60″); B. C., 35″ (n/—30″). Left ear, A. C., 10″ (n/—60″); B. C., 10″ (n/—30″). Politzer's acumeter, left ear, 2 feet. Galton whistle, right, 0.1; left, 0.2. Monochord, right, A. C., 20,000; B. C., 23,000. Left, A. C., 18,000; B. C., 20,000. Western Electric audiometer No. 2 shows hearing in right ear normal; left ear, loss 64 per cent. (Note—Right tube patent to politzerization, left is not.)

Turning tests: Right turning and left turning, 10 to 20 times a minute, produce neither nystagmus nor symptoms of vertigo.

Caloric test: Ten cc., 20 cc., and 50 cc. of cold water, at 50° F. (10° C.) do not produce on either side nystagmus or sensations of vertigo.

Conclusions.—There has been a fracture of the base, involving the left petrous bone; there was some immediate injury to the eighth nerve. Hearing to the spoken voice was completely and immediately lost on the left side; the left vestibular mechanism was upset. Compensation for the latter occurred very rapidly, and as a consequence the good ear as well as the involved ear will not react to either the caloric or turning tests. Hearing is moderately impaired in the left ear, while the right ear hears perfectly.

Case 7 (Dr. Stauffer's case).—William B., age 43. Chief complaint deafness and dizziness of two years' duration. One year ago he had to give up his work as a machinist on account of deafness, dizziness and dyspnea on exertion.

F. H.: No deafness in the family history; three children; none deaf or dumb.

S. H.: He chews a package of tobacco a day, does not use tea, coffee or liquor. He went through the war and has apparently developed a mild psychosis, which shows itself in a certain whinishness, in a spirit of introspection ("I cannot work, I am too sick"), and in an antagonism to those about him. There has been no history of discharging ears, and he cannot account for the onset of his deafness. He had typhoid in '98, no history of malaria or rheumatism (has not been on quinin or salicylates).

Examination: Fracture of the nose and external deflection to the right; right nostril stenosed, S shaped deflection (10 years ago he had his nose injured). Bad dental infection. Subacute congestion of the fauces and posterior wall; no tonsillar infection apparent, no pus in nose or throat; ear drums retracted, no scars, no discharge, canals dirty. Widened palpebral fissures, no spontaneous nystagmus, no nystagmus on eversion, reflexes normal, associated ocular movements become incoordinate on rapidly moving eyes from one extreme to the other. The eyes stagger and a slight rotatory nystagmus is pro-

duced, but there is no vertigo. "He cannot go upstairs without holding on with one hand, because of dizziness." There is no past pointing; Romberg is negative. Pupils are equal; react to light but not to accommodation. Wassermann (?).

Turning tests: To ten turns in either direction with the head erect, 90° forward and back, there is no reaction of nystagmus and no vertigo (except that there is a very fleeting sensation and a questionable transitory nystagmus from the horizontal canals). There is no past pointing after turning.

Caloric test: 30, 60 and 90 cc., 60° F., give no nystagmus or symptoms from either ear.

Hearing has not been carefully tested, as the patient is restless under examination and shows irritability and fatigue. It is noticed, however, that he hears spoken voice on both sides well when he does not let his attention relax. There are many evidences of attention fatigue.

Conclusions.—The patient is syphilitic, probably, though no confirmative Wassermann has been taken. His vestibular reactions are gone to both the caloric and turning tests, and yet the hearing is not markedly affected. In such a case a central pathology is more likely than a peripheral one. His history is typical of many of the cases—periods of vestibular upset (vertigo attacks), at which time the labyrinth will react to some degree to tests, and when there will be spontaneous nystagmus. In the intervals the labyrinth will be apparently dead. Complete accommodation takes place over a period of some months, the vertigo attacks come less and less frequently, and finally cease altogether.

Case 8.—James McGlade, age 71. Chief complaint is deafness. Gives a history of having received a blow on the left ear 20 years ago, followed by an ear discharge and complete deafness. Occasional severe rheumatic symptoms. Family history negative as to deafness. His hearing must have recovered to some degree, for on November 27, 1923, he reports that he could hear well in the right ear before two months ago. There is a ringing and blowing tinnitus. He has never suffered from any dizziness. Wassermann is negative. Knee jerks are markedly exaggerated, Romberg shows a slight swaying. No abnormality in facial muscles; the tongue protrudes slightly to the left; associated eye movements are normal. Nose: There

is an extreme deflection of the septum to the right; the mucosa is congested; tonsils are small and congested; both drums are slightly retracted, otherwise are normal; the tubes inflate easily.

Hearing examination shows that this old man is not grossly deaf in either ear at this time. There is considerable fault in his attention sphere.

Fork C256: Right, 3:12 inches; 90:120 seconds; B. C., 50:70 seconds. Left, 1:12 inches; 50:120 seconds; B. C., 60:70 seconds. Weber referred to the right.

Caloric test, 50 cc., 15° C.: Right ear, N. appears in 32 seconds, lasting 99 seconds. Left ear, N. appears in 42 seconds, lasting 90 seconds. There is no vertigo during the tests.

Conclusions.—There is slight bilateral nerve deafness, in addition to the loss of hearing that comes with age. The left side is more deaf than the right. There is no evidence that the injury of 20 years ago has had any effect. The labyrinth reacts slightly subnormally. The findings suggest a peripheral cause (low grade eighth nerve atrophy).

Case 9.—Montague, F., age 72. Chief complaint is deafness of long standing. There is a history of an initial sore when 19 years of age. He has been taking antiluetic treatment, off and on, all his life. A vocalist by trade, his musical abilities and appreciations are now greatly restricted by his nerve deafness. He has never had vertigo.

Examination shows a large perforation of the septum, a degenerated, atrophic nasal mucosa, nasal polyps, laryngeal thickening (fibrosis of the vocal cords), hoarseness; bilateral eustachian tube stenosis, relieved by bouginage, which seemed to improve the hearing and tinnitus. Drums appear normal.

Hearing tests: Fork C256 (B. C., 30; A. C., 100). Weber referred to the left. Rinne, minus on the right; plus-minus on the left. B. C., 25:30 on right, 35:30 on left. A. C., 15:100 on right (but he hears the high overtone, not the C^3); 30:100 on the left. Hartmann's 1046 heard better on the right than left; Hartmann's 256 heard better on the left than right. C64 fork heard on left, not on right. Galton 4.0 right, 5.0 left.

Voice tests: Threshold for spoken voice, 4 feet right, 2 feet left. Errors 36:100 R. E., 42:100 L. E. Omissions 0 right, 7 left. Monochord, A. C., 10.000 right, 6.000 left.

Caloric test, 50 cc., 60° F. (15° C.): Right appears in 35 seconds, lasts 85 seconds, no vertigo. Left appears in 45 seconds, lasts 25 seconds, no vertigo.

Past points below with the right hand moving in the horizontal plane; past points to the left with the left hand moving in the vertical plane.

Conclusions.—Both the hearing and the static labyrinth are affected in the luetic eighth nerve involvement. Hearing on the right is more affected; the vestibular branch is more affected on the left. On neither side is the involvement of the vestibular branch marked.

Case 10.—Ethel M., age 42, has had a cholesteatomatous discharge in the right ear for some years. There is considerable discharge in the right ear for some years. There is considerable drum destruction and at times polyps have been removed. She has never until recently been bothered with vertigo. The left ear is normal.

Hearing: Fork B488 (B. C., 30; A. C., 60)—Right ear, B. C., 10:30, A. C., 20:60; left ear, B. C., 30:30, A. C. 60:60. Galton, right ear, 0.9. Hartman C128 and C256 not heard on right; others heard. Weber referred to the left. Monochord, right ear, A. C., 13,000; B. C., 21,000. Left ear, A. C., 21,000; B. C., 21,000.

Caloric test: Right ear (20 cc., 50° F.), nystagmus appears in 40 seconds, lasts 60 seconds. Left ear (10 cc., 56° F.), nystagmus appears in 10 seconds, lasts 60 seconds

Conclusions.—There is catarrhal deafness on the right side; it is of severe grade and is apparently encroaching on the inner ear. The vestibular reaction is less acute on the diseased side, although at acute stages or periods, the reverse might be found. The patient in this instance was examined when there were no symptoms of vertigo. The finding on the left side shows a very alert labyrinth.

Case 11.—S. G. B., age 63, complains of a tumor of the right orbit, which displaces the eyeball upward and outward. Dizziness bothers him considerably, and has been constant for two weeks. Cerumen was removed from both ears and the drums found normal. Monochord and fork testings show a well advanced nerve deafness, but voice hearing is much better than these tests would suggest. Wassermann negative. The

knee jerks are gone, there is an Argyll Robertson pupil, and pupils are unequal. There is no other evidence of cranial nerve involvement. Romberg is negative and there is no ataxia. He is chronically constipated. Admits an initial sore in his youth. Has never been on antiluetic treatment. Blood pressure 165-100. At his first appearance, February 17, 1924, 10 cc. of cold water at 50° F. produces in each ear in 12 seconds a nystagmus that lasts one minute, and makes him feel "just like the attacks of vertigo that he has." Nux aggravated his symptoms; mixed treatment improved his symptoms. March 25, 1924. Dizziness has passed off.

Caloric reaction: Left ear, 10 cc., 48° F., no reaction; 25 cc., 48° F., nystagmus in 30 seconds, lasting 40 seconds. Right ear, 5 cc. 48° F., no reaction; 10 cc., 48° F., no reaction; 25 cc., 48° F., nystagmus in 30 seconds, lasting 20 seconds. March 28, 1924. Right ear, 10 cc., 54° F., no reaction; 20 cc., 54° F., no reaction; 30 cc., 54° F., no reaction; 90 cc., 54° F., nystagmus in 1 minute, lasting 1 minute (slight sensation of dizziness).

Conclusions.—He is an old syphilitic, with nerve deafness, and with an involvement of the vestibular branch. On the occasion of his first visit he had a labyrinth that reacted acutely (10 cc. produced a nystagmus in 12 seconds that lasted 1 minute). Subsequent to that time his reactions were all subnormal; such findings are typical.

Case 12.—Miss Julia A., age 45, school teacher, complains of frequent bilious attacks which are accompanied by dizziness. Previous medical history: As a child she had frequent severe earaches; no history of discharging ears. She recalls that she never could dance, or use the swing, or go on the merry-go-round, for these things would make her "deathly sick and dizzy." All her life she has been subject to "dizzy-sick" spells; sometimes she would be in bed from them two weeks at a time. During these attacks she would feel perfectly well if she stayed quiet, but became so dizzy on rising that she had to go back to bed. She would have no appetite, and if she ate she would vomit. "The attacks generally came on in sleep." Lying flat on the back made the dizziness worse, or, if she was recovering and had no dizziness, would bring back the attack. She has been bothered all her life with chronic constipation

and has had all sorts of treatment for it. Fifteen years ago she had a severe attack of neuritis, in the right side, about the waistline ("it was not shingles"), lasted three months; the pains came on sharply and lasted an hour at a time. She has been told by a physician who saw her in her last attack of vertigo and "biliousness" that she had gallstones; he recommended operation.

She describes her last attack as follows: "Felt nauseated on waking for three mornings in succession. On the particular morning of the attack she got up, took a light breakfast and kept feeling better as the day grew on. She took a nap between 2 and 4, got up feeling well, played cards, went to supper at 6, feeling well and not in the least apprehensive. She took one swallow of cold water and had the sensation of having something stirred up in the stomach. At the meal there was food that she liked particularly well, and she made up her mind she would eat it. She became so nauseated and dizzy that she had to leave the table and go upstairs, holding on to the balustrade. She vomited six times a white frothy fluid ("not green bile as usually"), but some lettuce salad eaten at noontime came up undigested. Went to bed, was dizzy in bed, could not move her head, had to close eyes; if she lay flat on her back she got worse. She was in bed a week with this attack.

Mental confusion or mental upset has always disturbed her hearing and her sense of balance. She can, however, stand going up on high places and look down without symptoms. "Elevators are apt to make her a little squalmish."

Examination: Ears, retraction of both drums marked, but the drums are excessively flaccid to the Siegel otoscope. This apparent contradiction is explained by the possibility of adhesions drawing in the hammer handle, for the malleus itself does not move with pneumomassage; the drum about the handle flaps like a sail. Neither tube is patent to politzerization.

Hearing: Monochord, right ear, A. C., 12,000; B. C., 19,000. Left ear, A. C., 13,000; B. C., 12,000. Galton, right ear, 1.5: left ear, 1.7. Weber referred to the left, even with the fork on the right mastoid. Fork B488, right ear, A. C., 30:60; B. C., 0:30. Left ear, A. C., 43:60; B. C., 25:30.

"Hearing worse on fatigue and worse in damp weather." Notices no difficulty in speech interpretation. Pressure changes

in the E. A. M.: Right ear, sound decreased on pressure, increased on making a vacuum in the E. A. M. Left ear, sound increased on pressure, decreased on making a vacuum in E. A. M. Audiometer No. 2 shows the following percentage losses for the various pitches:

Pitches	64	128	256	512	1024	2048	4096
Right ear	100	62	54	52	46	31	30
Left ear	49	45	41	28	27	23	13

Caloric threshold test, water at 55° F. (12.5° C.): Right ear, 5 cc., no reaction, no subjective symptoms; 10 cc., no reaction, no subjective symptoms; 15 cc., no reaction, no subjective symptoms; 20 cc., no reaction, slight subjective sensation of dizziness on looking to extreme right or left; 35 cc., no reaction, slight subjective sensation of dizziness on looking to extreme right or left; 65 cc., no reaction, slight subjective sensation of dizziness on looking to extreme right or left. Left ear, 10 cc., reaction in 30 seconds, lasting for 30 seconds, Q. C. to right, with subjective symptoms of dizziness.

Past pointing: Vertical past pointing to right with right hand, error of two inches; otherwise past pointing is negative. Wassermann negative; Romberg negative; associated eye movements normal; pupils equal, react normally; cranial nerves, except the eighth, are normal; tendon reflexes normal; blood count and urinalysis are normal.

Conclusions.—There is a bilateral deafness of moderate grade, worse on the right than on the left for the middle range of hearing. The monochord shows the left ear quite deaf to high tones. The physical findings of relaxed drums, hammer handles drawn in and tubes closed, point to a long standing conductive deafness, with probably a certain amount of metaplasia of the tympanic mucosa. The right static labyrinth is apparently dead, yet it is probable that the pathology on this side "lights up" at the times of the dizzy spells, and if the caloric reactions were taken at these times there would be found some degree of response, certainly greater than the nearly negative findings here shown.

The most singular feature of the case is the normal reaction of the left labyrinth. The vestibular symptoms have been

present since childhood; according to the experience of other cases, there should have developed an accommodation that would have given a left labyrinth incapable of stimulation. However, there is a normally reacting left side.

It is my opinion that the bilious attacks are a part of the symptom complex of the vestibular pathology, that the vomiting and stomach distress are a result of the static confusion. The importance of a differentiation is seen when it is realized that this patient might have been operated upon for gallstones. It is regrettable that the findings do not suggest anything toward the relief of symptoms and suggest still less as to exactly picturing the pathology affecting the vestibular mechanism. It is worth while noting, however, that the fluid extract of colchicum improved her hearing to a noticeable degree, relieved a mild tinnitus, and improved the constipation. To date she has had no recurrence of the attacks. The colchicum was given on the assumption of an element of neuritis in the case.

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Beck believes that as inflammation of the middle ear increases, the time between the starting of syringing and the appearance of nystagmus decreases. This time phase depends upon extralabyrinthine conditions (Krproff). The duration of the nystagmus on the contrary is independent of the degree of inflammation and a rather constant value, so that it is supposed to be called forth by intra-labyrinthine or central conditions.

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Boeters uses a caloric threshold method.

3. Brűnings: Beiträge zur Theorie, Methodik und Klinik der kalorimetrischer Functionsprűfung des Bogengang-apparates. Theory, Methods and Clinical Findings with the Calorimetric Functional Test of the Semicircular Canals. Zeitschrift für Ohrenheilkunde, 1911, vol. 63, p. 20.

Brűnings describes his otocalorimeter and his otogomometer; the latter is used for maintaining a fixed position of the head. For the normal functional test Brűnings has the patient's eyes everted from the tested ear 50 degrees. Irritability is measured by the amount of water (of 27 degrees Centigrade) that it takes to produce a nystagmus.

4. Demetriades, Th. D., and Mayer, Ph.: Zur kalorischen Labyrinthprüfung mit Minimalreitzen. On the Caloric Labyrinthine Test with Minimal Stimuli. Monatsschrift für Ohrenheilkunde, 1922, LVI, p. 654.

5. Eckert, A.: Ist der Nystagmus bei kalorisch Schwach- und Stark-reizen physikalisch oder physiologisch bedingt? Is the Nystagmus of the Caloric Reaction with Weak and Strong Stimuli Physical or Physiologic? Zeitschrift für Hals-, Nasen- und Ohrenkrankheiten, 1922, vol. 2, p. 165.

Kobrak, in a monograph recently published on the static functions of the human body, has collected the experimental observations made with his weak-irritation method, together with theoretical considerations. Eckert criticises Kobrak's methods and theories, as well as Biehl's and Griessmann's, and prefers Barany's rinsing en masse method.

6. Gradenigo: Ueber die graphische Aufzeichnung des vestibularen Nystagmus. On the Graphic Registration of the Vestibular Nystagmus. Zentralblatt für Ohrenheilkunde, 1910, vol. 8, p. 504.

Gradenigo uses a single turning of the turning test to determine the threshold, analogous to the minimal caloric stimulation.

7. Grahe, K.: Beiträge zur kalorischen Auslösung der Vestibularreaktionen. Contribution to the Caloric Reaction. Passow's Beiträge, 1920, vol. 15, pp. 167-179.

Grahe confirms Kobrak's findings with minimal stimuli, and reports nystagmus, falling reactions and past pointing with as little as 5 cc. of cold water.

8. Krproff, J.: Quantitative Messung des kalorischen Nystagmus bei Labyrinthgesunden. Quantitative Measurement of Caloric Nystagmus in Normal Labyrinths. Passow's Beiträge, 1909, vol. 2, pp. 129-140.

Krproff determines his minimal stimulation by timing the douching, and notes in 127 normal cases that cold water at 30 degrees Centigrade will produce a nystagmus in between 15 and 45 seconds of douching time. He everts the patient's gaze away from the tested ear to insure an early appearance of the nystagmus. This nystagmus appears most quickly in complete destruction of the tympanic membrane, with labyrinthine changes of the epithelium, after radical operations; it appears most slowly in otitis externa and after simple operations. The beginning of the nystagmus is in all probability dependent on anatomic extralabyrinthine conditions—i. e., the conduction of heat and cold.

9. Michaelson: Beiträge zur kalorischen Funktionsprüfung des Vestibularapparates. Contribution to the Caloric Functional Test of the Vestibular Apparatus. Deutsche Zeitschrift für Nervenheilkunde, 1913, vol. 46, pp. 222-227.

Michaelson uses Brüning's calorimeter and reports cases of multiple sclerosis showing increased irritability.

10. Quix: Apparat zur Erregung des statischen Labrynthes. Apparatus for the Excitation of the Static Labyrinth. Twenty-first meeting of the German Otological Society in Hanover, May, 1912 (reported in Zeit. für Ohr., 1912, vol. 66, p. 167).

Quix presents a "Statokineter," or "canule a double courant," by which the quantity and temperature of water can be easily read off.

11. Spira, Josef: Ueber das Verhalten des gesunden Vestibularapparates bei einseitig Labrynthlosen nebst einigen Bemerkungen

über die quantitative Prüfung der vestibularen Erregbarkeit. On the Condition of the Sound Vestibular Apparatus in Patients Having but One Labyrinth, and Some Remarks on the Quantitative Test of Vestibular Irritability. Monatsschrift für Ohrenheilkunde, 1922, vol. 56, p. 611.

Spira believes that whatever may be the cause of the destruction of the labyrinth, the other ear may lose its irritability for caloric reactions. The cause of such phenomena is unknown, but probably central conditions are playing the chief part. There may also be a relation with Ruttin's compensation of the turning nystagmus. This takes in cases where the labyrinth has been destroyed for some time and where all nerve elements are dead. For both caloric and rotation nystagmus equal values are found in both ears in spite of there being but one labyrinth diseased.

12. Wojatschek, W.: Ueber genaue Messungen der Funktion des Vestibular-apparates und die Nystagmographie. Concerning the Exact Measurements of the Functions of the Vestibular Apparatus and Nystagmography. Arch. f. Ohrenheilkunde, 1909, vol. 79, p. 139.

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Report of 513 investigations in 131 cases with Brünings' otocalorimeter. The patients show an inconstant intensity of nystagmus; even normal ears in the same patient gives different results. . . . Acute inflammatory and catarrhal changes in the middle ear diminish the degree of reaction; chronic suppuration also diminishes irritability. (In speaking of the above diseased conditions the author refers only to them when they are limited to the middle ear. It is well known that acute inflammation of the inner ear increases vestibular irritability, and that chronic inner ear inflammation tends to diminish irritability.)

14. Zalewski, T.: Ueber den kalorischen Nystagmus auf Grund eigener Untersuchungen. Concerning Nystagmus Based on Experiments. Lidowski Tyg. Lek., 1914, vol. 104, pp. 70-72.

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VIII.

OBSERVATIONS CONCERNING THE RELATION OF ENDOCRINE CHANGES TO NASAL (SPHENO-PALATINE GANGLION NEUROSIS ("LOWER-HALF" HEADACHE).

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The relation of endocrine disturbance to nasal neurosis and headache has received some mention in medical literature. Headache of pituitary origin is described by Pardee[1] as bitemporal, very intense, "boring" in character. He reports a series of seven cases who were given pituitary extract with gratifying results. Glassberg,[2] Dalche[3] and Trimene[4] also describe a pituitary headache and report excellent results from the use of pituitary extract. Lawrence[5] reports an interesting case of pituitary insufficiency in which the predominating symptom was hyperesthetic rhinitis, completely controlled by continued administration of pituitary extract. Frank J. Novak[6] reports a type of hyperesthetic rhinitis, which he terms type C (in contradistinction to type A, hay fever and spring fever, and type B, hyperplastic ethmoid type). In these cases he claims to "effect, if not a cure, a complete cessation of symptoms," by the use of thyroid extract. At the time of the publication of his article (March, 1921) he believed that these cases showed a low metabolic rate, but in a recent communication Dr. Novak advises me that he now believes "that the metabolic rate is practically of no value as a test in many of these conditions," and "moreover, hyperesthetic rhinitis with a normal metabolic rate showed improvement of symptoms under thyroid therapy." W. L. Simpson[7] calls attention to Novak's article, and states he has found that "a very few cases of hyperesthetic rhinitis have a low metabolic rate and are relieved by the administration of thyroid extract." Mithoefer[8] in a recent article says that "There is another type of patient in whom the nasal mucosa is constantly in a state of chronic infiltration,

and who seeks medical aid for the relief of an incessant headache. A careful study of this patient shows that there are present symptoms of hypothyroidism. The insufficiency of the thyroid is usually of the benign type without any external evidence of myxedema. The cellular protoplasm of these patients is constantly overloaded with mucin and fat, and the headache is probably due to an infiltration of nerve cells and of the meninges. We have observed cases of this type in which headaches disappeared after the use of small doses of thyroid extract over a long period. Some of these patients state that, while taking the thyroid, the insomnia from which they had long suffered disappeared, and that the constipation was overcome. The intestinal stasis, which is a characteristic symptom of hypothyroidism, is probably due to muscular infiltration of the intestinal wall." According to Timme: "Disturbances in the gonadal series of organs are reflected in other glandular elements, notably the pituitary and the thyroid. When the testicles degenerate as a result of an orchitis secondary to parotiditis, the pituitary body undergoes enlargement. With this enlargement mental confusion is a frequent concomitant; such patients are also prone to have migrainous headaches of pituitary type from causes which earlier in life were inoperative in this respect. Curiously, in psychosexual excitement, headaches may arise intratemporally with all earmarks of pituitary disturbance; and at least two cases have come under the writer's notice in which acromegalic symptoms following the onset of headaches, produced during intense and acute psychosexual stress, pointed directly to the pituitary body as the source of the headache. The headaches that come periodically with the menses, or rather preceding the menstrual period, have been called "ovarian" by some authors. The real explanation, however, the writer believed to be as follows: Towards the end of the interval between menstrual periods, the secretion of the corpus luteum diminishes. This secretion is antagonistic to the thyroid secretion and hence its diminution marks the onset of a relative hyperthyroid state. This determines, first, a hyperthyroid headache; and secondly, through stimulation of the pituitary by the excessive thyroid activity, a pituitary headache. Either one may take on the characteristics of migraine."

It is not within the scope of this paper to discuss various endocrine theories, or to refer to the many reflex phenomena connecting the nose and organs of regeneration. The writer wishes to record some observations in certain cases which have come under this notice. These observations seem to show a definite relation of hypoendocrinism in the production of nasal ganglion neuroses. Sluder[10] describes this type of headache as a pain beginning "at the root of the nose, in and about the eye, the upper jaw and teeth, and extending backward to the temple and about the zygoma to the ear, making earache; emphasized at the mastoid, but always severest at a point 5 cm. back of that; thence reaching backward by way of the occiput and neck, it may extend to the shoulder blade and shoulder (less often to the axilla and breast), and in severe attacks to the arm, forearm, hand, and even to the finger tips." This headache in no way resembles the pain attributed to pituitary engorgement.

Chas. B. Williams[11] reports three cases of nasal ganglion neurosis with endocrine imbalance, which were benefited by endocrine medication.

Everyone who has studied Sluder's syndrome has noticed that in many cases the "explosion" (headache) bears a definite relation to the menstrual period. For example, the following case is cited:

Mrs. M. has been under my care for some time for attacks of severe "lower half" headaches. These attacks occur a few days preceding menstruation. The pain starts in the left upper jaw and teeth and radiates back to the ear and behind the mastoid, and sometimes to the neck and shoulder. The pain is promptly relieved by cocainization of the nasal (sphenopalatine) ganglion. She gives a definite history of postethmoiditis in the past.

This history is in no way unusual, but has some bearing on the analysis of the cases reported below.

Case 1.—Mrs. J. B., aged 26, consulted me November, 1922, for headaches, dizziness and feeling of stuffiness in both ears. The headaches were not confined to either side and had been recurrent for five years. The pain always started in the temple, on one or the other side, radiated backward to the mastoid, and thence to occiput and back of neck and occasionally to the

shoulder blades. These attacks had increased in frequency and were more usual on the right side. There was a stuffy feeling in the ears (the hearing was actually normal) which did not seem to be relieved to any extent by inflation. There was the usual history of colds in the past, but no attack which one could point to as the origin of the trouble. (I have often found this to be so in cases of long standing.) To my surprise, on repeated examinations no pathology was evident in the nose. Each nasal ganglion was repeatedly cocainized with a saturated solution of cocain, with transitory, if any, relief. Her feeling of dizziness was not explained by the vestibular tests, as the latter conformed with the normal. As her eyes had been examined by a competent ophthalmologist, they were eliminated as a cause of headache.

Upon questioning her as to the possible relationship of her headaches and her menstrual period, it was learned that five years previously, shortly before the beginning of her headaches, both of her ovaries had been removed, because of some cystic condition. She was referred to an internist for a general physical examination. He found nothing of importance other than an excess of fat and lowered basic metabolic rate. Patient was given corpora lutea (soluble extract) by hypodermic, and thyroid, gr. ¼, and ovarian substance, gr. 5, by mouth. Rapid loss of weight, 20 pounds, followed.

Following the endocrine therapy, her symptomatology rapidly cleared up—headaches ceased altogether. The stuffiness of the ears was the last symptom to disappear.

Case 2.—Mrs. E. H., aged 37, presented herself for examination March 20, 1924. She complained of a pressing sensation in the right temple, extending to the vertex and to and behind the right ear, with actual pain about 5 cm. back of the ear. There was no history of nasal or postnasal discharge. She also complained of tinnitus in the right ear and great nervousness. She claimed that the whole condition arose just five months previously, just prior to an ovarectomy. She has been much worse since this operation. Examination showed no pathology in the nose or throat, except for apparent sagging of the palate on the left side. Cocainization of the right nasal ganglion gave only temporary relief from these symptoms. Basic metabolism was decreased 28 per cent. The following

treatment was instituted: A hypodermic injection of one ampoule of corpora lutea every third day, and by mouth a capsule containing ovarian substance, gr. V, and thyroid extract, gr. 1/6, three times a day.

She has been under treatment too short a time to arrive at a definite conclusion in regard to benefit. Her physician reports her as improved.

Case 3.—Mrs. F. T., aged 38, consulted me April 21, 1923, for a severe left sided "lower half" headache. She presented all classical symptoms of the Sluder syndrome. The pain started with an attack gradually increasing in severity and becoming almost unbearable. It began about the root of the nose, in the left eye, extended to the temple, zygoma, ear, postmastoid, occiput, neck and left shoulder. (She stated that in some attacks the pain had extended to the finger tips of the left hand.) The pain was quickly relieved by cocainization of the left nasal ganglion. In a second attack about one week later, the pain complex was identical with the first attack, and there was added also the sympathetic syndrome, vasomotor and secretory; severe sneezing, intense congestion of the nasal mucous membrane, oversecretion of mucus, redness of the external nose, injection of the conjunctiva, and marked photophobia. All these symptoms were relieved by cocainization of the ganglion. On May 7, 1922, a careful examination was made at the office, and no nasal pathology was demonstrable. This examination coincided with the results of a previous examination made by another rhinologist. She did, however, give the following interesting history:

Some three years previously, after the birth of her last child, her headaches commenced. At the same time she developed other symptoms which caused her obstetrician to give her corpora lutea. Her headaches ceased completely for over one and one-half years while she submitted to regular administration of endocrines. For the past nine months she had received treatment only spasmodically, or had neglected it altogether. The headaches had returned and had increased in frequency. Her "explosions" usually occurred a few days before her menstruation. Her basic metabolism was tested just two days before her period and was found to be decreased 47 per cent. She again resumed treatment with corpora lutea. She has been

under treatment with endocrines for over one year, and has had only two attacks of headache during this time, on each occasion when she had neglected treatment.

Case 4.—Mrs. W. S., aged 52, has been under my care for headaches for the past seven years. She gave a definite history of acute postethmoiditis bilateral some fifteen years previously. Following that attack, she began to have "lower half" headaches, most marked on the left side, becoming less and less frequent, until she entered the menopause. The headaches then increased in severity and frequency. Her headaches were typical of nasal ganglion neurosis; usually starting in the left temple, radiating backward to postmastoid and neck, shoulder, arm, and frequently to the finger tips. They were always promptly relieved by cocainization of the ganglion. She refused injection of the ganglion. Other nervous phenomena were marked, and four years ago she spent some time in a sanitarium in the East, where she was given thyroid and corpora lutea with marked improvement; the headaches were lessened in severity and frequency. She was a very difficult patient, however, and often neglected her treatment. The menopause is now complete and her headaches no longer occur.

It would be unwise to draw any definite conclusions from so scanty material. But these cases give food for thought. In the first instance, we have an example of "lower half" headache which was not affected by cocainization of the nasal ganglion. Several hypotheses are possible. In the first place, the ganglion may have been rendered susceptible to irritation by previous acute postethmoid sphenoiditis, and since the irritation due to metabolic changes was so constant and pronounced, the ganglion was resistant to treatment. Or it may be that previous infection is lacking, and that infiltration of the nerve cells with mucin and fat caused the neuralgia. Or that pressure of the surrounding tissues due to infiltration would produce this phenomena. (It will be recalled that the patient exhibited excessive and unnatural fat, and lost twenty pounds in weight rapidly after commencement of therapy.)

Sluder[10] advances the following theory in explanation of intractable cases: "If the cells of the nasal ganglion are as resistant as those of the semilunar ganglion, it explains how they may for some time resist in an inflamed environment;

but when once attacked they may in the same manner resist treatment. So it might be explained that intractable vidian neuralgia is not unrelated to intractable vasomotor phenomena, inasmuch as they are often associated and are alike resistant to treatment." Another reason for failure in local treatment may be found in one of the hypotheses advanced above. However, only the observation of a number of such cases, and further study of endocrines in their relation to nerve ganglia, will be of value in arriving at a definite conclusion.

Case 2 closely resembles Case 1, but not sufficient time has elapsed to draw any conclusion.

The third and fourth cases present a different type. In the first two, the attacks had reached a frequency that was almost constant. In the third and fourth the individuals had periodic attacks, and the attacks could be relieved by treatment. The neurosis seemed to have a definite relation to the menstrual period, as a rule, and a markedly low metabolic rate was established. Sluder calls attention to an observation of Buch that "the sympathetic in health was not especially sensitive, but that it becomes very sensitive if the animal be worried or the nerve becomes congested. These facts readily explained how these cases may relapse from fatigue, anxiety, apprehension, fright, anger or slight systemic toxemias, insomuch as the environment of the ganglion and the nerve tissues may remain slightly inflamed for almost any length of time, and pass under the generic term catarrh."

Since so many individuals show phenomena due to endocrine changes, and since Sluder's syndrome is unusual in such cases, it seems only logical to conclude that, in the cases reported above, the environment of the ganglion and nerve tissues, or the ganglion and nerve tissues themselves, have remained slightly inflamed from some forgotten infection, and have been stirred up by irritation produced by endocrine changes. In the first two the metabolic change was constant, due to surgical menopause, and in the last two the irritation was intermittent, due to periodic changes in internal secretion.

In conclusion, I can only add that these patients were definitely relieved by endocrine therapy, and that absence or diminution of ovarian secretion seemed to be a positive factor in producing their symptoms.

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IX.

TREATMENT OF FIBROMAS OF THE NASOPHARYNX: REPORT OF THIRTY-TWO CASES.*

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The type of treatment to be selected for new growths is that which will give the best end results in each particular case. It is usually necessary to combine methods of treatment in order to obtain the best results; we believe, however, that certain of these methods are of major importance, and the others are of minor importance in the treatment of a given case.

Tumors of the nose, throat and mouth may be classified in three groups for the employment of the major form of treatment: (1) Tumors best suited to surgical procedures, (2) tumors best suited to cautery or diathermy, and (3) tumors best suited to radium and X-ray treatment. In Groups 1 and 2, radium or X-ray is used in conjunction with the other methods. In our experience, fibromas of the nasopharynx should be placed in Group 3. Radium is the treatment of choice.

During the last fourteen years, 1910 to 1923 inclusive, we have examined at the Mayo Clinic thirty-two patients with fibromas of the nasopharynx. This group includes only the hard fibromas. The ages of the patients varied from ten to thirty-one years, the average being eighteen and one-half years. Twenty-nine (90.6 per cent) were less than twenty-five years of age at the time of examination, and three (9.3 per cent) were between twenty-seven and thirty-one years. The activity of the tumor seemed to be directly related to the age of the

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†Alonso collected from literature thirteen cases of fibromas in the nasopharynx in women.

patient, although the fact that operations had been performed in many instances made it difficult to determine this point. Such tumors are more common in the male; there were twenty-eight males and four females in our series.† The average age of the males was nineteen years, and that of the females fifteen years.

The duration of symptoms previous to examination was from two months to six years. Five patients had had symptoms for less than one year, nine for more than two years.

PREVIOUS OPERATION AND TREATMENT.

Twenty-three of the thirty-two patients in our series had been operated on from one to twelve times before their examination in the clinic. The average number of operations was between three and four. Two patients said that they had had "multiple" operations; these operations were not included in the average. Five patients had not been treated previously. Treatment had consisted of internal medication, the application of caustics, fulguration and X-ray. Two patients had been treated with radium.

HISTORY, CLINICAL FINDINGS AND DIAGNOSIS.

Fibromas of the nasopharynx present a typical clinical picture; they have a definite life cycle, starting to grow at about puberty, and continuing are active until the age of about 25 years. The tumor is amenable to treatment in older patients, since there seems to be a spontaneous retrogression after the age of 23 or 25 years. The tumor, which is hard, may be pedunculated or have a broad sessile base. It may have several attachments due to ulceration, and may fill the greater part of the nasopharynx. In ten of our series the tumor was confined to the nasopharynx alone, and in thirteen it had extended to both nostrils. The antrum was involved on one side in two cases and the palate in three. The tumor had apparently originated in the right side of the nasopharynx in eighteen cases, in the left side in nine, in the median line in three, and in one case the point of origin was not determined.

Fibromas of the nasopharynx are microscopically benign and clinically malignant. This type of tumor does not metastasize, but may, owing to its location and extension into the skull,

cause death. In some of the earlier cases, tissue was removed for microscopic examination; this proved to be fibroma, fibroangioma or fibromyxoma. In a few cases the specimen was reported inflammatory, due to the reaction from repeated previous operations and treatments. In the last four years no attempt was made to obtain tissue, on account of the bleeding which accompanied such procedure, and the diagnosis was based on this history, the hardness of the tumor, the age of the patient and the clinical picture.

TREATMENT.

The surgical treatment of fibromas of the nasopharynx is usually attended with a high mortality and frequent recurrences, necessitating repeated operations. The severe hemorrhage which accompanies such operations makes them hazardous. Goldsmith has recently reviewed the literature carefully and completely, and reported two cases. Wojatschek, in a recent contribution, reported eight cases in which operation was performed by the transpalatal or transmaxillary route; although he had no immediate operative mortality, several patients had recurrences and one died subsequently of the disease. The condition of these patients was not observed for more than one year postoperatively. Kobylinski, in a series of ten cases, reported three deaths due to hemorrhage. Dabney reported one case recently of a boy in whom the condition had almost entirely cleared up following one radium treatment. Crowe and Baylor reported four cases treated with radium. Two of their patients are well and the others are improved. Alsonso reported the case of a woman, aged forty-five years, who was well three years after operation and radium treatment.

The patients in our series, who were treated previous to our use of radium in these cases (1910-1915), were operated on by the avulsion method, both through the nose and the nasopharynx. During these years eight patients were observed; five were operated on and two died; we were unable to trace the other three patients; three patients were examined only, and not treated.

From 1915 to January 1, 1924, twenty-four patients with fibromas of the nasopharynx were examined. Twenty-three were treated with radium. The treatment was applied by three

methods. In the first, or early cases, a T shape lead applicator, with a 50 mg. tube of radium in the trough of the T, was held in various positions against the tumor in the nasopharynx. The original dose was usually a 50 mg. tube for from ten to fifteen hours. It was difficult to apply the radium accurately in this manner, and there was severe reaction in the structures around the tumor. In two cases the palate was perforated. Such complications are now prevented by protecting the posterior surface of the palate with a lead retractor made of 2 mm. of lead, covered with a rubber finger cot, and by more accurate dosage.

In the second group of cases, steel points, containing the radium emanation or the element, were inserted directly into the tumor. Three or four points were usually inserted, depending on the size of the tumor, about the same amount of radium being used as in the initial dose.

In the third group of cases emanation seeds, averaging 0.5 to 1 millicurie each, were implanted directly into the tumor, the number depending on the size of the tumor. This method has been used in recent cases only. Such treatments are repeated in from six weeks to two or three months, depending on the reaction and the result of the previous treatment. The dosage of the secondary treatments varies greatly, depending on the progress of the case. The number of applications required, in the cases now cleared up, varied from two to nine, averaging between five and six. The length of time our patients were under treatment before the condition cleared entirely varied on account of the distance some of them lived from the clinic and the difficulty in returning; the average length of time was fourteen and two-tenths months. Several patients had been operated on elsewhere, with much bleeding. The radium seems to have controlled this immediately. The crusting and scabbing secondary to radium treatment may be cared for symptomatically by the use of oil sprays, potassium iodid internally, and so forth. Table 3 demonstrates that treatment, and not the natural regression of the tumor, is the important factor in the cure of a patient. Eleven (73.3 per cent) of patients were less than twenty-three years of age when cured, and four (26.6 per cent) more than twenty-three years of age when cured.

SUMMARY.

Radium is the treatment of choice for fibromas of the nasopharynx. With its use the usual operative mortality in these cases is eliminated. In most of our cases the tumor cleared up entirely, and in the others it was held in check during its period of activity.

TABLE 1.

END RESULTS.

1910 to 1915—Patients treated before the use of radium	8
1915 to 1923—Patients treated after the use of radium	24
Patients examined before the use of radium	8
Patients operated on	5
Operative deaths	2
Not traced	3
Patients examined only	3
Patients examined after the use of radium	24
Patients cured	15
Patients under treatment	4*
Patients examined only	1
Patients not traced	4†

TABLE 2.

FIFTEEN PATIENTS CURED.

Patients well from 7 to 8 years	2
Patients well from 6 to 7 years	0
Patients well from 5 to 6 years	2
Patients well from 4 to 5 years	1
Patients well from 3 to 4 years	3
Patients well from 2 to 3 years	3
Patients well from 1 to 2 years	4

*The four patients under treatment at the present time registered at the clinic in 1923.

†The four patients who were not traced improved during treatment.

TABLE 3.

AGE OF FIFTEEN PATIENTS WHEN CURED.

Eleven (73.3 per cent):

13 years	1	18 years	1
14 years	1	20 years	1
15 years	2	21 years	2
17 years	1	22 years	2

Four (26.6 per cent):

24 years	1	28 years	1
27 years	1	32 years	1

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X.

DIAGNOSTIC LIMITATIONS IN LARYNGOLOGY.

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I realize that perfection in diagnosis is unattainable, but the impression has been growing upon me that we take our failures and our responsibilities in laryngeal diagnosis too lightly. This seems to be particularly true with regard to that triad, syphilis, tuberculosis and carcinoma. In whatever way we may elect to explain the situation, the fact remains that too many cases of laryngeal tuberculosis have reached an advanced stage before the diagnosis is made, and too many laryngeal cancers have passed the operable period before the condition is recognized.

Some of our limitations are due to a lack of fundamental facts, which will, no doubt, be in part supplied by the general progress of medicine; some will inevitably remain as a perpetual legacy until the millennium, but there remains a certain percentage of our failures which could be minimized by a more consistent and understanding partnership with the other departments of medicine.

Much might be gained by a closer attention to the diagnostic field which lies out of reach of the laryngeal mirror. An effort to make a diagnosis by inspection alone is an error made by all of us too frequently. If the modern trend toward divorce is detrimental to the social fabric, it is equally true that the present day separation between the specialties and general medicine is not an unmixed blessing. This is generally recognized in theory and quite as generally ignored in practice.

Laryngology, like other fields of medicine, has benefited greatly by the development of the Wassermann reaction, but, like other branches, it suffers occasionally from misplaced confidence. A proper degree of caution in accepting the report of a test which is conceded to have an unavoidable percentage of error will minimize the damage.

The literature abounds with splendid articles on the differential diagnosis of tuberculosis of the larynx, but this condition has been treated as a local lesion for so many years that the pulmonary basis is still frequently ignored. Diagnosis cannot be determined without a thorough knowledge of the clinical and pathologic phases of the disease, general as well as local, and if this knowledge is not possessed by the laryngologist it must be supplied elsewhere.

It may seem trite to say that a negative Wassermann in the presence of a pulmonary lesion does not exclude syphilis or prove a tuberculous laryngitis, yet this knowledge is not always applied. It is equally true that we are occasionally beguiled by a positive Wassermann into accepting a cursory, or at least inefficient chest examination, thereby risking confusion between lues and tuberculosis. Having not infrequently accepted the apparently obvious diagnosis, only to find that an unusual and unexpected combination of these lesions existed, I am more and more inclined to approach the entire subject of laryngeal diagnosis from the negative side. Because a patient with pulmonary tuberculosis develops hoarseness, it does not necessarily follow that the hoarseness is due to tuberculosis, nor does a positive Wassermann exclude malignancy. Tuberculosis, syphilis and carcinoma show a wide divergence of incidence, and while the law of probability may serve to point the way to a diagnosis, it is a weak stick to put much weight upon.

Even commonly accepted views are not always above reproach. In advanced laryngology, the teaching that superficial ulceration of a spreading character is due to tuberculosis and that the clear cut, punched out ulcer is syphilis, has no place. Certain signs which may be called pathognomonic may impress the student in his first years of medicine, but such teachings react against scientific diagnosis in the end.

I am assuming, of course, that few of us will ever attain perfection in distinguishing these lesions by inspection alone. If this be granted, it is at once apparent that we must depend for diagnostic aid on other men, whose results we must accept, but whose methods and authority we cannot always vouch for. Jackson[1] has well said, "Clinical work is not and never can be ideally perfect. We are human and our patients are human."

Not infrequently a case is referred by a physician who reports the chest negative, and because of ethics his report is accepted without question, thereby denying the patient the proper general examination. Errors in technic must be allowed for. Too much reliance on a single negative examination may lead to a wrong diagnosis. No one can be right all of the time.

As cases carry more weight than mere generalities, I would like to report two instances where the Wassermann was of vital importance, one from the literature and one of my own.

"Harmon Smith's[2] case of a woman, 48, hoarseness, six to seven weeks' duration, increased shortness of breath and choking sensation, no difficulty in swallowing. Examination: Right arytenoid, part of interarytenoid space and aryepiglottic fold on right side showed typical pyriform edema, with congested and reddened base. Right cord restricted in movement, left side uninvolved. Palpation, mass very hard. Puncture made into apex of swelling. It was found to be a hard resisting tumorous structure. Wassermann made immediately, and, while waiting for report, a second puncture was made with the thought of finding pus. Wassermann four plus. Mass disappearing with salvarsan, mercury and iodid."

This case is reported to demonstrate the ease with which improper diagnosis can be made and how essential it is to arrive at a definite conclusion by the exclusion method rather than by reliance on one's vision."

One cannot but speculate on what the outcome of such a case might have been had the first Wassermann been negative and had the case been in the hands of one less skilled and who used less care in arriving at a diagnosis than Dr. Smith.

The following case of my own will demonstrate how even such elements as luck would play an important part in diagnosis. A man, aged 37, was referred to me by a well known laryngologist, who used every care in arriving at a diagnosis. The chief complaint and symptom was hoarseness, which followed an attack of influenza. In referring the case, the doctor said he had made a provisional diagnosis of tuberculous laryngitis because of the appearance, but stated clearly that he did not believe in primary tuberculosis of the larynx, and that it had been impossible to demonstrate tuberculosis elsewhere in the body. He had a Wassermann done, with a negative re-

port. He had on various occasions cauterized some ulcerations in the larynx without response, and therefore advised him to make a change of climate. The patient weighed 178 pounds; had no rise of pulse or temperature. His voice was very hoarse; had no expectoration. There was a right sided infiltrative lesion of the larynx, making almost a shelflike projection and binding the false and true cords together throughout their entire length. I explained to him that even though he had been thoroughly examined and every test had been done, these should all be repeated. As on the previous occasion, no tuberculosis could be demonstrated, but the Wassermann was four plus. The larynx recovered promptly under antisyphilitic treatment. Again I speculate as to what procedure I would have followed had the second Wassermann been negative.

Perhaps my best excuse for touching upon carcinoma in any definite way is the fact that the present teachings on this subject are by no means clear.

Harmon Smith[3] states that, "Laryngologists with the best and most earnest intentions vary in their methods of procedure in laryngeal carcinoma." Let me quote a few of the conflicting views of those who should speak with authority on a single phase of the problem—biopsy.

Mackenty[4] thinks biopsy is robbing us of our powers of observation and is open to criticism here, as it is in general surgery, where it is resorted to only in exceptional instances. He has been forced to employ it in only a few instances among forty-eight cases.

Harmon Smith[5] favors biopsy and emphasizes that for definite conclusions of any value it is necessary to remove a piece of the tumor extending well into its basal structure. Views expressed in the discussion of Fraser's paper, before the section of laryngology, Royal Society of Medicine, are as follows:

Fraser[6] employed it in every case after the patient's consent to operation had been obtained.

Tilley[7] thinks it would be better to make a mistake in the diagnosis than to interfere with the growth before operation.

St. Clair Thompson[8] approves of preliminary biopsy if a satisfactory piece can be obtained.

Dundas Grant[9] has seen cases in which the diagnosis could not be made without biopsy and does not think it a detriment to the patient's chances.

Jobson Horne[10] opposes removal beforehand, but thinks it should be done and examined at time of operation and the course to be pursued determined upon after receiving the tissue report.

Syme[11] would remove a piece in every case.

Opinions thus seem to be about equally divided as to whether we should regard biopsy as a legitimate aid to diagnosis or a criminal offense. If we decide to use it we must still bear in mind that, like other diagnostic weapons, it occasionally proves to have a very dull edge.

In the discussions at last year's meeting, Sluder[12] mentions two cases in point. In one of these, two eminent pathologists reported malignancy, but the patient gave no evidence of such involvement ten years later. In another of Sluder's cases, two bits of tissue removed at different times were reported negative, while subsequent operation revealed a cancer.

We can, all of us, subscribe, however, to Delavan's[14] expression, "Improvements in the methods of diagnosis which would admit of a clearer vision of the probable course of the growth, would add greatly to the possibilities of successful treatment."

Broder's findings[13] may be a step toward this goal. It would seem from Broder's investigations in typing and grouping cases of laryngeal carcinoma, that much may be learned of prognostic value from a study of tissue sections. These cases showing only a small proportion of differentiated epithelial cells have a poor outlook. Incidentally, these conclusions may throw some light on the conflicting views on biopsy, which should not be blamed for operative failure where the microscopic picture shows a high grade of malignancy.

In presenting this very loosely drawn bill of particulars on our diagnostic limitations, I am clearly aware of the existence of that uncanny diagnostic sixth sense, which does not depend upon the teachings or the literature for its inspiration. Delavan[14] speaks of a "subconscious reasoning based upon long experience of many closely observed cases. The beginner does not have the instinct for diagnosis. The man of long practice

may acquire it." I can only remind you, however, that the great majority of patients must, of necessity, pass through hands which do not possess this gift.

In conclusion, I can but reiterate my belief that the limitations and responsibilities of diagnosis in this field are not sufficiently recognized by the average laryngologist. To this might be appended the by no means unique statement that this specialty suffers by too great an isolation from the parent stem.

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FERGUSON BUILDING.

XI.

SOME POINTS IN THE OPERATIVE SURGERY OF THE MASTOID WHICH ASSURE THE QUICKEST POSSIBLE TIME IN HEALING.

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The mastoid operation, performed for the relief and cure of acute mastoiditis, was, less than a half century ago, classed among the hazardous operations in surgery. The process of repair and the complete healing of the wound were usually long drawn out, often requiring as long as two or three months, and sometimes becoming chronic discharging wounds. Even much more recently complete healing after this operation often required four to six weeks, even in favorable cases.

At present this operation may be performed practically without danger, and the average time of healing is probably something less than three weeks. Formerly the postoperative period, the period of healing, was one of distress to the patient, the change of dressing was painful and the long period of wearing some sort of bandage was embarrassing. Today all this is changed—the healing is rapid, the dressings comparatively painless and bandages are necessary but for a few days. These happy results have been brought about by a better knowledge of the pathology involved, early and positive diagnosis, and by the development of a greatly improved, one might perhaps say, an almost perfected technic for this class of mastoid operation.

In the vast majority of all acute mastoid infections the disease is limited to the middle ear and mastoid cells, with perhaps a continuance of the original and causative infection in the nose, pharynx and nasopharynx. No vital center is involved in simple, uncomplicated mastoiditis. If, therefore, the disease is diagnosed early, as it now easily may be, and before any

extension to adjoining and more dangerous environmental structures occurs, the process of eradicating the ailment by surgical means is one of the simplest of all surgery, and therefore should be, and is, one of the safest of all operative procedures. In short, surgery of the mastoid for acute masoiditis has, for the writer at least, attained the standard of a well established and thoroughly settled surgical procedure.

Quick and satisfactory recovery from this operation depends largely upon the proper and thorough execution of certain points in surgical technic. These in my own practice have been evolved and adopted after an experience of more than twenty-five years in mastoid surgery, during which several thousand mastoid operations have been performed. It is not presumed that this technic differs materially from that of others who have likewise noted the results of the various plans which have been from time to time employed, and who have finally adopted a plan based upon an experience which is highly satisfactory to themselves.

The principal points in this operation, the strict observance of which give quickest healing and safety, are:

1.—The Preparation of the Field of Operation.—My early teaching was, that since the mastoid operation is performed in a septic field, great care in the sterilization of the mastoid is not wholly essential. It probably is not wholly essential to ultimate success, but thoroughness in sterilizing the area to be operated on is entirely necessary to the quickest healing, to comfortable convalescence and certainly to absolute safety of the patient. The same care should, therefore, be taken to prepare the field of operation and to protect it from a nonsterile environment during the operation that would be considered adequate in case the operation were to be done on the brain or in the abdominal cavity. For the purpose of sterilization I use the ether, iodin and alcohol sequence, and have no desire to change.

2. Exposure of the Affected Osseous Surfaces.—While not, of course, an advocate of undue or unnecessary exposure of the underlying mastoid bone, I have little patience with efforts at operation made through inadequate incisions, for it is undoubtedly true that large wounds will heal quickly if all the disease is removed, whereas small wounds never heal if some

seriously affected part is left behind. An adequate exposure of one's work is the best insurance that all of the disease will be discovered and safely removed.

Formerly I extended the incision some distance into the neck below the mastoid tip, for the purpose of facilitating detachment of muscles. This, I believe, is a mistake, except in case of Bezold's abscess. The incision should be but slightly beyond the tip toward the neck and only just far enough to enable the operator to detach such portions of attached muscle as may be actually necessary. Some years ago the muscles were very completely detached from the mastoid tip in all cases before opening the mastoid antrum and cells, and, therefore, before it could be known whether or not extensive detachment would be helpful. There are many cases in which but little detachment is desirable, and in these, if complete separation of the muscle is made, unnecessary pathways of infection are opened, more postoperative pain than necessary is invited and prolonged healing often results.

3. The Extent and Manner of Bone Removal.—The mastoid antrum should, I believe, be opened in every case, and every suspicious mastoid cell ought to be eradicated. The osseous wound does better, heals quicker and more certainly if left a little rough. It is an error in technic to hone or polish the osseous cavity of a mastoid wound, for to do so delays to some extent the final healing of the wound. The posterior meatal wall should in every case be cut away partially, almost to the tympanic ring, much after the manner of the Heath operation. This sharp osseous septum, which formerly was left standing between the external meatus and the mastoid cavity, has no function and is undoubtedly a hindrance to quick healing. When cut away it allows the soft structures to drop into the wound on closing, to partially fill the wound, and thus very greatly to favor rapid and complete healing. The removal of this useless obstructive osseous structure will, I estimate, enable cure to occur at least a week earlier than if the same is left standing. This removal of the postmeatal wall does not end in any asymmetry of the two ears after healing is complete.

4. Another important point in the technic of securing the quickest possible time in healing is found in the management

of the mastoid tip. Formerly, as did others, I removed it completely. I now believe to remove the tip is a mistake in nearly every instance, since to do so requires completely detaching the muscles attached to it, which is usually entirely unnecessary and therefore an error of technic. There can be no argument concerning the fact that all infected mastoid cells located in the tip should be completely eradicated, but this can be done, and should be done, without molesting the osseous shell which forms the floor of the tip and internal wall of the tip. If this hard shell is left in situ the neecssity for muscle detachment is wholly avoided or reduced to a minimum, drainage will be equally good, while postoperative soreness and stiffness of the neck due to muscle infection will almost certainly be avoided. In most cases no good reason whatever can be assigned for completely removing the tip of the mastoid. To leave it allows all the neck muscles to function, and, because these muscles are not cut, muscle infection is avoided, and another point in securing quick healing is established.

5. Drainage and Packing.—It was formerly thought necessary to pack all mastoid wounds until healing was complete. "Secure healing from the bottom" was the dictum. This plan usually insured safety and ultimate healing. However, as a result of this method, the cure was often long delayed, an unsightly, depressed scar remained, and the several weeks or months of daily change of the packing was more or less painful. This procedure, fortunately, has largely fallen into disuse. Healing by granulation from the bottom of the wound was a slow and entirely unnecessary process, but for the reason that the mastoid operation had been performed in the midst of sepsis this technic was long thought best and safest, and was reluctantly given up by many in great fear of the supposed evil consequences likely to follow any other method.

In this connection mention may be made of the benefits derived from allowing the patient to be out of bed soon after the mastoid operation. A large percentage of such patients have been ambulatory up to the day of operation. To confine these to the bed for any undue period, as was formerly done, is not necessary to safety and often retards progress to early cure. Therefore, unless the case is already prostrate from the disease which caused the mastoid affection, it is a wise plan to

allow the patient to sit up and to be about the room on the second or third day after operation.

6. Employment of the modified blood clot dressing was a long step forward to quick healing in mastoid operations. Infections of the clot sometimes occurred, but it was in due time observed that serious infection, or at least serious consequences following infection of the clot, could be avoided in nearly every case by strict observance during the operation of all the points of surgical technic herein discussed, and especially those dealing with the preparation of the wound, thoroughness in removal of all infected tissues, and especially of the insertion of a proper drain for the first twenty-four hours.

No blood clot could organize and remain a wholesome factor in healing in the presence of infection. It is with this thought in mind that the field of operation must be made thoroughly sterile before beginning the operation; gloves must be worn, and every other precaution followed throughout that will of a certainty secure sustained sterility. Any suppurating or infected cell that may be overlooked at operation will always break down the blood clot, set up danger and slow down or completely arrest the healing process. Hence every point of even minor detail must receive attention during the operation. An item of value in preparing all mastoid wounds to receive the blood clot is that of attention to all minute bone chips and grit from the use of the chisel and hand burr. Small bone chips are easily overlooked, and bone grit from the burr is, of course, not easily seen. Both or either must, however, be completely removed if the blood clot is to be used and quick healing is to be expected. I am of the opinion that attention to the simple detail of completely removing this operative debris is of prime importance in securing quick, certain and painless healing of the operated mastoid. My plan, before finishing the wound, is to drop into the mastoid cavity a dram or more of pure hydrogen peroxid, and, after a few moments, to syringe the same away with sterile water of a temperature at about 110° F. Neither the peroxid nor water are used with the primary thought of sterilizing the wound, but rather to blanche the bone and to make more certain the removal of all osseous debris. The foaming peroxid seems to entangle the osseous particles, which may then readily be washed away

by means of the hot water. The bone is whitened, and the small bleeding is checked by this procedure, the resulting clean wound giving the operator a better opportunity to make a final inspection of the cavity for any pathology that may have been overlooked. At this inspection I have sometimes discovered an unopened cell or area of diseased bone which, if left behind, would of a certainty have caused delayed healing.

When the wound is known to be thoroughly operated and sterile, a small rubber tissue drain is inserted into the open cavity of the mastoid antrum. I no longer use a wick or cigarette drain, believing that the simple rubber tissue secures better drainage and is less inviting to outside infection. This drainage tissue is removed at the first dressing and no other is inserted. Insertion of the drain into the antrum is best because it then drains away any suppurative product from the middle ear. Mere insertion of the drain into the mastoid wound may invite infection of the clot from infectious products remaining in the ear.

After placing the drain as stated, the soft structures of the ear and mastoid are allowed to fall into the mastoid cavity, and any dead space remaining to fill with blood. If the posterior meatal osseous wall has been cut away sufficiently, as already described, and the mastoid tip dealt with as previously indicated, but little dead space will remain after the tissues have fallen into place; therefore, but little blood clot is required to complete the filling in of the operated cavity. Indeed there often seems to be necessary but a thin layer of clot; only just enough to insure what corresponds to a union by first intention between the bone and overlying soft tissues, and therefore it is not uncommon to find healing practically complete in ten days.

7. The Sutures.—Placing the sutures would seem too simple a procedure to mention. However, there are points connected with it which must be observed if comfort and quick healing are to follow. In cases where the soft structures of the mastoid are thickened by infiltration, caution is necessary not to tie the sutures tightly, since more swelling than is already present usually follows the operation, and if tension is great on the sutures both pain and suture abscess will result. Suture abscess may infect the underlying blood clot and other healing

parts, and this would of a certainty greatly delay complete healing of the wound. Four sutures of catgut are used, two of which are usually removed on the second day and the remaining ones on the fourth day. Sutures of silk, silkworm or other nonresisting material are never so comfortable or safe, and never are conducive to as quick healing as that secured from the more elastic catgut. To secure perfect, painless healing, healing without a scar, requires of course a perfect coaptation of the opposing lips of the wound. It has been argued that partial opposition is all that is necessary, since if the edges are left partially separated better drainage is secured. Experience does not bear out this contention.

Following the above plan of operation for acute mastoiditis, there is usually no suppuration from the wound. The drainage is small and consists of serum. The drain tissue is removed on the first or second day and none other replaced. The external auditory canal is mopped dry, if necessary, with alcohol and the line of sutures over the mastoid wound are swabbed over with the alcohol. This very simple attention is all that is necessary in a very large percentage of cases, those that follow severe infections as well as the mild cases doing about equally well. Even when the dura has been uncovered, or where an extradural abscess has been evacuated, the wound is closed over as above described, and the after treatment is carried out along the same simple lines, for experience has shown that in the more extensive cases, where the dura is exposed, or where infection seems menacing, do best under the simple plan of treatment. Of course, in cases where there is a reasonable question as to whether or not all the products of infection have been removed, the operator will wisely omit the lower sutures, pack the wound and keep its depth under observation. The cases in which this plan is wise are few, for should the wound do badly, in any case the difficulty is shown in pain and rise of temperature, in which case it is not a serious or difficult matter to release the sutures, open the wound widely and remove the source of trouble. The wound may then be packed and otherwise treated as was formerly done when packing was the rule in every case.

XII.

FRONTAL LOBE ABSCESS: SUBSEQUENT HISTORY OF A CASE PREVIOUSLY REPORTED.

By Samuel Salinger, M. D.,

Chicago.

At the January meeting of the Chicago Laryngological and Otological Society (Annals of Otology, Rhinology and Laryngology, June, 1924), I presented a young man of 18, who had made a recovery from a left frontal lobe abscess following a frontoethmoid sinusitis. At the time the case was shown, the patient still had a small unhealed fistula at the site of operation and had passed through three months of complete wellbeing since the abscess was evacuated.

Subsequently a plastic operation was performed closing the frontal wound, with very satisfactory cosmetic results. The boy was kept in the hospital for another month, during which time his condition was absolutely normal insofar as we were able to determine. He was discharged from the hospital as completely cured on May 2, 1924.

On May 15th, the patient was brought back to the hospital with a history of severe headache beginning four days previously. He vomited twice on the day of admission, the emesis being accompanied by marked nausea. He appeared to be acutely ill, his face having an anxious look and his color being an ashy gray. At the site of the old frontal scar above the brow was a pulsating, slightly herniated mass. The wound was completely closed. A probe pushed through the medial end of this scar and withdrawn was followed by a few drops of pus. There was some rigidity of the neck, the Kernig was positive and Babinski negative.

The spinal fluid was under pressure and gave a cell count of 450, globulin plus. No organisms on smear or culture. There was a bilateral neuroretinitis, with marked dilatation of the retinal veins. Swelling of the nerveheads was not so marked. His eyes were closed, but he would open them when

requested. The pupils were equal and reacted to light and accommodation. He had a spontaneous past pointing with the left hand about four inches to the right. Sense of smell was intact. The patient kept moaning continually and moving his jaws. Temperature, 100.4° F.; pulse 116; respiration 24.

It was evident that we had to deal with a recurrence of the old abscess or a new abscess in another location. Consequently he was immediately reoperated. The old L incision was opened. A herniation of the scar over the old abscess cavity presented with marked pulsation. A few drops of pus oozed from under the edge of the bony defect which, when followed by a probe, led to a cavity. No further pus appeared. The hernia was incised and the old collapsed cavity explored, but no pus was encountered. A brain searcher introduced in every direction to a depth of 4 cm. failed to reveal the secondary abscess. The wound was left open and the patient put back to bed.

On the following day, a further detailed neurologic examination failed to elicit any new or focalizing symptoms. The patient survived another twenty-four hours and then passed out. Autopsy revealed the following:

"An evacuated abscess of the anterior portion of the left frontal lobe. A secondary abscess of the posterior portion of the left frontal lobe with local acute hemorrhagic encephalitis. (This abscess was about the size of a hazelnut, seemed to be well encapsulated, did not communicate with the original abscess and posteriorly came within 5 mm. of the tip of the anterior horn.) Absent anterior and posterior and medial walls of left frontal sinus; old exenteration of left anterior ethmoid cells; missing left lacrimal bone; left nasofrontal fistula; edema of the brain; turbid spinal fluid; hyperemia of the vessels of the leptomeninges; gaping operative wound of the left nasofrontal area; left enophthalmos; moderate saddle deformity of the nose; partially resected right fifth costal cartilage; fibrous union of two nasal cartilaginous transplants; healed operative wound of the nose; rubber drainage tube entering anterior abscess of frontal lobe; fatty changes of the myocardium; hyperemia and edema of the lungs; bilateral fibrous pleuritis; hyperplasia of left diaphragmatic lymph glands; fenestration of the aortic leaflets."

The interesting features about this case were:

1. The long period of quiescence between the appearance of the first and second abscesses.

2. Entire absence of symptoms during the interim.

3. The sudden recrudescence with symptoms of meningo-encephalitis.

4. The failure to locate the second abscess in spite of symptoms pointing to its location and a most careful search.

5. The marked spontaneous past pointing of the arm on the affected side in the opposite direction. Attention has been called to this symptom in connection with frontal lobe lesions by Oscar Beck, Voss, Bárány and Eagleton, the explanation of it being as yet undetermined.

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XIII.

JUGULAR THROMBOSIS FOLLOWING TONSILLECTOMY.

By Dr. Charles Goodman, F. A. C. S.,

New York.

In a case of primary jugular thrombosis due to tonsil infection, presented by the author before the surgical section of the New York Academy of Medicine, at a stated meeting held January 5th, 1917, the interesting features are the direct infection of the jugular by the lymphatic route from the tonsils and the pharynx, instead of the usual primary involvment of the sinus, and a secondary extension to the jugular complicating mastoiditis.

In previous literature, Long's case of sore throat with streptococcic invasion of the internal jugular vein and subsequent thrombi most nearly approaches this one.

Barnes, in a compilation of reports in systemic infections secondary to follicular tonsillitis involving the pharyngeal wall, mentions no direct infection of jugular vein as a complication.

We may conclude that the probable bacterial agent was streptococcus, since it is the most frequent factor in acute infectious processes in the mouth and throat (Braun) as well as in thrombosis.

It is interesting to quote here the explanation given by Libman and Celler of the negative blood cultures they obtained in a number of cases when sinus thrombosis of infectous origin was present: "We are inclined to believe that there may be early no bacteremia, that the bacteria may then be found perhaps in increasing numbers, and that it is possible for the bacteria to decrease in number and even to disappear before there has been any operative interference.

It is also possible that in certain cases there is below the infected clot an obturating, noninfected clot which prevents the bacteria from entering the circulation. We can also imag-

ine the possibility of there being an infected clot which completely closes the vessel. Another possible explanation is that in the course of the infection the blood may acquire a higher bactericidal power."

By analogy the same arguments might be applied to the negative blood cultures in our cases of jugular thrombosis we have just reported.

Valtan reports one case of thrombosis of the bulb of the internal jugular, with ligation and evacuation of the thrombus. Valtan remarks that the disease generally sets in without fever and then the pyemic picture with the classic fever curve appears, followed by a stupor, with compression phenomena of the ninth, tenth and eleventh nerves. To decide the nature of the fever, lumbar puncture is advisable. There is usually also a very painful edema at the posterior margin of the mastoid; with percussion on the skull there is an easy recognition of the seat of the pain in the middle fossa and the lateral sinus. The thrombus generally begins from above; when it begins from below (jugular vein) it means that the infection of the tympanic membrane has proceeded by contiguity through the mastoid to the bulb. In both cases there is a hardened cord along the neck representing the thrombosed jugular and the adjacent infiltrated connective tissue, which extends below the mastoid along the anterior margin of the sternocleidomastoid muscle, but this local symptom may be absent, indicating the formation of the thrombosis has not extended so as to invade the entire jugular vein. At times upon palpation only a small lump can be made out towards the top. Locally with ausculation the special bruit of the jugular may be detected, but one must always press the stethoscope against the vein to avoid hearing the sound of the carotid. Often there is an exaggerated irritability of the movements of the head, especially the inclination of the head towards the diseased side, as the patient endeavors to avoid the pain provoked by the pressure on the diseased jugular region. In deglutition pain on the diseased side occurs.

Mosher reports a case of deep cervical abscess. He explored the neck for pus, and uncovered the internal jugular vein for possible thrombosis. The carotid and the pneumogastric nerve were laid bare for some two inches. The vein

could not be recognized, over the nerve there was a thickened strip of tissue indistinguishable from thrombosed vein or thickened vessel sheath. At this point in the operation the patient stopped breathing. The routine measures usually employed in such emergencies were without success. After the patient's death a thorough exploration of the wound failed to identify the vein. An inch of the thickened tissue which lay over the nerve was excised.

Later the pathologist stated that this was probably disorganized and thrombosed vein. No autopsy was allowed. Mosher concluded that the vital structure in the neck from the standpoint of infection is the internal vein. In deep cervical abscess the vein is always exposed to the danger of infection. In such cases where there occur chills or a septic temperature, the vein should be laid bare, and its condition determined. If it shows phlebitis only the vein should be tied; if it is thrombosed, it should be dissected out. Pus about the internal jugular vein is analogous to pus in contact with the lateral sinus. We have always considered the infection of the lateral sinus or the internal jugular vein must come from the mastoid. Infection, however, can reach the vein from the tonsil just as readily as from the middle ear.

Page reports a case of septic thrombosis of the jugular bulb in a girl who had a myringotomy. Exploration of the sigmoid sinus after the temperature rose to 107.5, revealed scant bleeding from the bulb, but no clot in the vein, and no apparent change in its wall. The internal jugular was resected, and the lower portion of the sigmoid wall cut away. There were formations of septic thrombi in the sigmoid, and lateral sinuses, which were removed and iodoform gauze inserted. The gauze was removed on the second day, and there was an uninterrupted recovery.

Cordier and Rollet reported a case of endopericarditis, and of asystolie in a man of thirty. The autopsy showed a phlebitis of the brachiocephalic vein and thrombus involving also the internal jugular as well as the subclavian. Forty-eight hours before death the patient showed exophthalmus, pupillary paresis, and ptosis and diplopia; all of which phenomena are explained very simply by the simple hypothesis of stasis.

We must admit, however, as in intermittent exophthalmus, that this stasis is favored by a special condition of the ophthalmic veins, which become more readily dilated.

On May 4th of this year, I presented a second case of jugular thrombosis, following tonsillectomy, at the New York Academy of Medicine. The patient in this case is a doctor, a refugee from Armenia, who is pursuing his studies here. He had been suffering with indefinite pains about the shoulders and the arms. These pains were attributed by his physician to a probable focal infection of the tonsils. This was emphasized because his physician found that his left tonsil contained a large crypt filled with purulent material. His physician performed a tonsillectomy under local anesthesia for him last December. Four days after the tonsillectomy the patient felt pain and discomfort on the left side of his neck. He was advised to make hot applications, which he did for two days, but the swelling in the neck increased. He could not swallow, and he developed a high fever with chills. He was admitted to my service in the hospital when he had a temperature of 103½. He was somewhat cyanotic, he had some difficulty in breathing and considerable difficulty in swallowing, and appeared to be very septic. Foul smelling pus welled up from the pharynx, but just from what point could not be determined, because he could hardly open his mouth. The left side of his neck was the seat of an extensive brawny swelling. My diagnosis was cellulitis of the neck with jugular thrombosis. He was immediately taken to the operating room, and under anesthesia I made a rapid ligation of the jugular vein, which was found thrombosed. I then incised the phlegmon and found an extensive necrosis of the deep fascia of the neck, including the carotid sheath. Upon introducing my finger into the wound the tip entered the pharynx. Both the wounds were left open. Blood culture taken at the time proved sterile. Cultures taken from the throat showed staphylococci and diphtheroid bacilli. His blood count showed 28,000 whites with 82 per cent polynuclears. Food taken was found to leak through the communication of the pharynx into the wound. We turned the patient on his right side and fed him fluids through a tube. He made a gradual recovery and now feels perfectly well.

The arteries are known to show a remarkable resistance to infections contiguous to them. Veins also have considerable resistance, but to a very much less degree. In this case I believe that the infection was a lymphatic extension from the pharynx, and traveled through the walls of the vein.

Patient A. B., male, 29 years old. Had tonsils removed under novocain anesthesia on Friday, January 11, 1924. The left tonsil contained an abscess cavity. The evening of the same day the patient bled considerably, but the hemorrhage was controlled by application of thromboplastin. Five days later the patient had a rise of temperature and complained of acute pain in the left side of his neck, for which an ice bag was applied. He was admitted to the surgical service of Beth Israel Hospital on January 18th. He presented a brawny swelling on the left side of his neck, extending from the mastoid down to the clavicle. There was tenderness upon pressure over the anterior border of the sternocleidomastoid. His temperature was 104.4 and he appeared very ill and septic.

Diagnosis: Jugular thrombosis (left); cellulitis of neck.

Operation: As soon as the general anesthesia was commenced the patient developed pronounced dyspnea, with substernal retraction. I attempted to introduce a tube into the larynx but was unsuccessful, so I performed a low tracheotomy. The jugular vein, after being exposed and found to be collapsed, was ligated with catgut ligatures. The neck was then incised and a large quantity of foul smelling pus was evacuated from the cavity which led up to the wall of the pharynx. The deep fascia of the neck was necrotic. Three days following operation his temperature dropped to 101 and the tracheotomy tube was removed. The general condition of the patient shows a decided improvement, and he is now well on the road to recovery.

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XIV.

A NASAL FIBROMA—SURGICAL REMOVAL—RECURRENCE—TREATED BY RADIATION.*

By Benjamin D. Parish, M. D., and

George E. Pfahler, M. D.,

Philadelphia.

Nasopharyngeal fibromas are unique in that they are benign tumors, in the pathologicoanatomic sense, but their course is malignant because of rapid growth. The rare occurrence of nasal fibroma is demonstrated by the fact that at the Basel Clinic only eleven cases have been observed in twenty-six years. They nearly always have an intrapharyngeal attachment and in all cases recurrences ensued one or more months after operative treatment. The literature also shows that under operative treatment one or more recurrences generally preceded complete permanent cure, and that in nearly all the cases cited unusually profuse hemorrhages occurred. These tumors are distinctly most prevalent during adolescence and after the age of twenty-five years spontaneous regression generally occurs.

This report is of a boy, S. F., age 14, who was referred to me for examination on June 8, 1921, because of certain symptoms of nasal obstruction and with the statement that he always seemed to have a cold in the head. His previous history was that he had had measles, chickenpox, whooping cough and his appendix had been removed. Also his adenoids had been removed in 1917. With the postnasal mirror a large smooth rounded mass could be seen extending well to the posterior pharyngeal wall and obstructing any view of the right eustachian tube. Anterior rhinoscopy showed a pinkish, white mass pressing against the septum in the right nostril and extending almost to the floor of the nose and completely hiding the middle turbinate and ethmoid region. The septum was rapidly being pushed toward the left, and symptoms referable to press-

*Presented before the Section on Laryngology of the College of Physicians of Philadelphia, January 16, 1924.

ure upon the eustachian tube were beginning to manifest themselves. There was considerable bleeding on probing, and the region within the nares showed areas of necrosis.

A small area of growth was snared off and examined by Dr. Case and the report of an angiomatous fibroma, benign, was made.

The patient was admitted to St. Agnes Hospital on June 20th for operation.

Having in mind the angiomatous structure, after consulting with Dr. George Dorrance, it was considered advisable to have the latter ligate both external carotids before attempting removal of the growth.

The question of the best way to approach the growth was considered. It was very desirous not to make any external scar or large deformity such as usually results from a Moure operation or any other external method. It was hoped that the growth had a pedunculated base, though it was impossible to determine this from examination except under ether.

Under ether anesthesia the growth was attacked postnasally with a snare and anteriorly with Luc and Bruning forceps. When sufficient amount of tissue had been removed, with a long periosteal elevator, an endeavor was made to separate the base, which was found to be very broad and attached to the vault of the pharynx and anterior sphenoid wall. The total amount of growth removed was about equal to a good sized egg.

The ligation of the carotids rendered almost bloodless the field of operation. The case left the hospital ten days after admission. I considered I had removed all accessible tumor tissue possible, but in view of the usual history of recurrence the patient was asked to return for examination after his summer vacation. Three months after the removal of the growth examination showed a mass again encroaching upon the ethmoid region but not extending back into the pharynx.

The case was referred to Dr. Pfahler for his opinion as to the advisability of using radiation.

Plates were first taken which showed dense shadows extending into the ethmoids and also into the right antrum. The cell walls of the right ethmoid as well as the upper and inner wall of the maxillary sinus were destroyed or at least decalcified.

Such bone destruction, without any evidence of repair, is almost characteristic of new growth. The X-rays prior to operation, taken at St. Agnes Hospital, showed no involvement of the antrum or ethmoids. On September 16th, three radium needles containing 30 mgs. were inserted into the tumor in the right nares and left in place for six hours. From this date radiation or X-ray treatments were given once each week up to December 5th. There was a total of 3260 mg. hours of radiation applied at a distance varying from 2 mm. to 2 cm., according to the location of the application. In addition to this he had X-ray treatment applied over the anterior surface of the left side of the face Nov. 28, 1921, using 9-inch spark gap, 5 milliamperes, 20 minutes time, 6 mm. filter, at the distance of 30 cm. A similar dose was applied Dec. 5, 1921, in the right submaxillary region and again on Jan. 21, 1922, in the right submaxillary region, and Jan. 21, 1922, in the left submaxillary region.

The detailed treatment with radium was as follows: Sept. 16, 1921—30 mg., 3 radium needles, 6 h., inserted into tumor. Sept. 26, 1921—80 mg. and 1B and 1R, 1.5 cm., 3 h., Rt. zygomatic area. Oct. 3, 1921—50 mg., 5 St. N and 1B and 1R, 2 cm., 3 h., Ant. surface R. max. sinus. Oct. 10, 1921—80 mg., 8 St. N. and 1B and 1R, 1.5 cm., 3 h., R. side face. Oct. 17, 1921—80 mg., 8 St. N. and 1B and 1R, 2 cm., 3 h., over R. ant. surface face. Oct. 25, 1921—80 mg., 8 Sn and 1B and 1R, 2 cm., 3 h., R. temporal region; 50 mg., Gen and R, 3 h., R. nares. Oct. 31, 1921—80 mg., 8 Sn. and 1B and 1R, 1.5 cm., 3 h., Ant. surface R. max. Nov. 7, 1921—80 mg., 8 St N and 1B and 1R, 1.5 cm., 3 h., R. temporal region; 50 mg., Gold and 1R., 3 h., nares, R. Nov. 14, 1921—50 mg., Gold and R., 3 h., L. nares; 50 mg., 0.5S and 1B and 1R, 0.5 cm., 3 h., R. inner canthus. Nov. 21, 1921—50 mg., 0.5S and 1B, 6 cm., 3 h., R. inner canthus; 80 mg., 8 St. Nd. and 1B and 1R, 1.5 cm., 3 h., outside Rt. temp. region. Nov. 28, 1921—50 mg., Gold and R, 2 cm., 3 h., in rubber catheter, R. nares, post. one-third. Dec. 5, 1921—100 mg., 1.5 S. and 1B and 2R, 3 cm., 2 h., against soft palate; 50 mg., 5 N. and 1B and 2R, 1 cm., 2 h., R. temporal region.

On November 14th, two months after radiation had been started, Dr. Pfahler reported that the right maxillary sinus was entirely clear, though there still was some increased den-

sity in the right anterior ethmoid cells. Elsewhere the growth seemed to have been removed, and that it was entirely possible that even this density in the right anterior ethmoid cells might be due to fibrous tissue, but he believed that for the time being it had better be considered as a part of the disease. My examination at that time showed a much increased open area within the right nostril and the area of recurrence greatly diminished, very much more firm and giving greater resistance to probing.

During the month of March, 1922, while at boarding school away from the city, he had a marked case of facial erysipelas.

In April, 1922, I could discover no visible evidence of the tumor, though Dr. Pfahler reported that the plates still showed an excess of fibrous tissue in the right ethmoid region, but would not recommend any further treatment unless there was some evidence of redevelopment of the growth. On the next examination, May 1, 1922, the entire mucous membrane had become dry and atrophic on the right side, and the septal quadrilateral cartilage had necrosed and been absorbed. Whether the original pressure from the growth, the attack of erysipelas or the radium was the cause for this loss of the septum it is difficult to definitely state, but we believe that it most likely resulted from the radiation. My last examination in the fall of 1923 showed no evidence of any return of the growth. The nasal mucosa was secreting much better; the crusting was much less and the boy was in excellent health.

Quoting from M. Güssow, on the Genesis of the Nasopharyngeal Fibromas (Ztschr. f. Ohrenhlk., etc., 82:103, Wiesbaden, March, 1922): "It is a striking fact that nasal fibromas occur almost exclusively in males. No satisfactory explanation has so far been advanced for this. The theory of Bensch is that the growth of the bony skeleton of the skull comes earlier to a standstill in woman than in man. Another puzzling feature is the occurrence of the tumors between the ages of 10 and 25. All explanations of this peculiarity revolve around the assumption of an abnormality in the course of the development of the skull. Their origin is believed by Virchow to be cell collections dating from fetal life.

There is still a wide divergence of opinion in regard to the place where these tumors originate. The original site has not

been microscopically examined in any of the cases so far published. There is, however, a specimen at the Breslau Clinic which solves this problem of their source. The sphenooccipital synchondrosis is seen in the center of the specimen to be composed of normal cartilage cells and sharply walled off from the tumor lying beneath it, to which it bears no relation. This disproves Gegenbauer's theory, according to which the starting point of the development of the nasopharyngeal fibroma is in relation with a process of ossification of the synchondroses. The real point of origin is quite patent. It starts from the basilar fibrocartilage, the cartilaginous connective tissue capsule which covers the under surface of the occipital bone, the foramen lacerum anterius and the fissura petrooccipitalis, at the point of junction of the three pairs of bands coming each from the region of the atlas, from the fissura petrooccipitalis and from the region of the pterygopalatine fossa. The nasopharyngeal fibromas stand midway in respect of their growth between the malignant and the benign tumors. Their penetration in the bone is achieved by the young tumor tissue burrowing its way into it and causing tissue absorption in its immediate neighborhood. This zone stains less readily than bone substance, and as long as this zone cannot be surgically removed there will be a recurrence of the tumor."

Coenen, on the Genesis of Basal Fibroid, in the Arch. f. klin. Chir., Berlin, 121:197, Dec. 14, 1922, states that: "The pedicle of the tumor lies in the middle part of the base of the skull, from the ethmoid to the great occipital foramen, and laterally to the foramen lacerum anticum and to the pterygopalatine fossa. A choanal, a pterygomaxillary and a tubal type can be differentiated.

The therapeutic principle is to decrease the tumors from time to time by slight operations until the twentieth year is reached, after which the basal fibroid disappears of itself."

In an article on "Nasopharyngeal Fibromas," Pierre Sebileau, Ann. d. mal. de l'oreille, du larynx, etc., Paris, 42:553, June, 1923, states that: "Exenteration of these bleeding fibromas should not be undertaken until two points have been considered: (1) the establishment of a way of approach; (2) provision for conservative measures, such as continuous anesthesia

at a distance; thorough tamponment of the propharynx; the employment of measures to maintain the patency of the air passages. Exenteration by torsion is preferred whenever practicable; the transnasofacial route is the alternative. Total resection of the superior maxilla constitutes, in the opinion of the author, a surgical monstrosity. He considers the two rational procedures to be (1) prelateronasal rhinotomy, the simpler operation, devised by Huguier and popularized by Moure; and (2) sublateronasal rhinotomy, the buccal operation devised by Rouge and modified by Denker."

S. J. Crowe and John W. Baylor (Arch. Surg., 6:429, March, 1923) report on "Benign and Malignant Growths of the Nasopharynx and Their Treatment with Radium," as follows: "There is an extensive literature on the subject of nasal and nasopharyngeal growths, but very little has been written in regard to their treatment with radium. Radium as a therapeutic agent is far more dangerous than the Roentgen ray. The authors report forty-nine cases at length, giving age, sex, duration of symptoms, clinical findings, treatment and the end result.

Nasopharyngeal fibroma is a tumor that occurs in boys 10-20 years old; it is characteristic of the tumor that as it grows it becomes attached to soft parts in contact; this characteristic renders its surgical removal very hazardous because of uncontrollable hemorrhage. Conservative treatment is particularly indicated in this type of growth; four cases are presented. Angioma originating on the lateral wall of the nasal cavity is not such a rare condition as some have thought; the experience of the authors convinced them that surgical extirpation of such growths is an extremely hazardous procedure. One outstanding result of radium treatment is the high percentage of cures in vascular tumors; although they do not disappear after irradiation so rapidly as some other types, the end result is much better."

In an article by Studer, Schweiz, at Basel, May 25, 1922, the authors urge that in every case of nasopharyngeal fibroma, radium and Roentgen radiation should first be tried. If these fail, operative methods are indicated, especially the technic of Denker or Killian.

Summarizing the outstanding points of interest to my mind in this case are (1) The almost bloodless operative field after ligations of both external carotids; (2) No disagreeable symptoms arising after ligation; (3) The gratifying result obtained after Dr. Pfahler's treatment with radium and the X-rays; (4) The tumor tissues demonstrable in the ethmoid cells by means of the Roentgenogram, together with the destruction of the septa and wall of the antrum; and (5) The disappearance of the tumor tissues and the reappearance of the bony walls after the radiation treatment, as demonstrated in the Roentgenograms.

XV.

ORBITAL COMPLICATIONS OF SINUSITIS.*

By James Allen Patterson, M. D.,

Colorado Springs.

The purport of this article is to bring to your attention some conditions not altogether unusual but which may be overlooked if one's routine examinations happen to be too hasty or the viewpoint too limited. The cases to be described are those on the borderline of ophthalmology and rhinology.

Case 1.—Colored, age 50; attack of "grip" Nov. 30, 1915. Her physician, in spite of swelling of the right eye, treated her for neuralgia until December 23, when, following several nights of extreme pain, she came to my office. The eyelid was so swollen that I could barely open it. The swelling extended upward over the frontal bone to within 2 cm. of the hairy scalp; no fluctation was obtained. Under local anesthesia the anterior end of the middle turbinate body was removed; pus was immediately released. Before leaving the office the swelling of the eyelids lessened, and by the next day the swelling had disappeared from the forehead.

Case 2.—Married woman, age 34. March 27, 1923, came in with the right eye protruding and the skin of the upper and lower lid reddened. She had had the "flu" two weeks previous, since which time the right upper eyelid had been swelling and receding intermittently. Under dilatation and suction the pain, which had been intense, was relieved, to recur daily. Pus was obtained by suction. On April 7, I amputated the middle turbinate and enlarged the frontal duct. Discharge continued for several weeks, but subsided under vaccine and syringing of the frontal duct with neosilvol.

Case 3.—A girl of 16 had suffered pain in the left side of the face, accompanied by vomiting, for three days. The patient thought it was a severe headache. Gradually the left eye

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began protruding from the orbit. The attempt to relieve pain by dilatation and suction failed, although pus in small quantities was seen in the nose afterward. The next day, following a sleepless night from pain, the condition was alarming. Proptosis of the eye was greater and the eyeball so fixed in the orbit that she was unable to move it in any direction. The swelling extended above the brow, and under the brow was greatest near the inner canthus. It was red and brawny. She was immediately removed to the hospital, where under ether anesthesia the anterior ethmoids were exenterated and the frontonasal canal rasped. An incision was made through the skin at the upper inner angle of the orbit, it not being possible to do so under the eyelid. Pus followed the incision; its culture showed staphylococci, streptococci and pneumococci, from which a vaccine was made. The patient gradually improved. For days pus flowed so freely from the operated nostril that it was necessary to keep a little cotton in the vestibule and to replace it frequently. As the swelling of the eyelid lessened, diplopia was complained of (superior oblique paralysis). This subsided, but vaccine and other measures failing to stop the discharge and particularly because there remained a discharging sinus at the point of the incision, a Lothrop operation was done a month after the first operation. This healed the sinus, stopped the discharge, and the patient had no further trouble excepting once or twice she had discomfort and swelling of the eyelid from swimming and diving in a cold water pool, but by shrinking the tissues at the outlet of the frontonasal canal she was immediately relieved. The pain was apparently caused by an air vacuum or air empyema, as no fluid was ever released by this procedure.

Case 4.—This case I have been unable to follow, having seen it only once, but it was at first quite confusing. C. P., age 22, works in a lumber camp in the mountains and lives in a slab boarded cabin, lined with paper, which he says is warm and comfortable. A week ago he had headache over the right eye which was greater at noon. There is tenderness under this brow, but no nasal discharge. He has diplopia, greater about 3:00 p. m. A glance showed that he could not close his right eye within 4 or 5 mm. of the lower lid, the uncovered portion being greater to the temporal side.

Aside from the pain, which was not intense, he complained only of a "scum" that comes and goes over the right eye. Before investigating I presumed it was a case of Bell's palsy, but I found his vision in each eye 6/6, with normal eyegrounds. There is a paresis of the right inferior and internal rectus as well as involvement of the facial nerve.

Dilatation and suction brought away an enormous amount of mucopus from the right nostril.

Case 5.—A woman of 68, in apparent good health, complained that the right eye had been extremely painful, keeping her awake at night. It is now slightly proptosed, there being a suspicion of increased intraocular tension. She claims that this eye has never been as good as its fellow. The vision is 6/9. The optic nerve is round with a large cup, with steep overhanging edges above. Later the patient admitted she had been having pain intermittently for the past six or eight weeks, with a severe head cold and a light bronchitis. On suction of the right nostril considerable secretion was obtained, but its exact source was not then determined. This treatment was continued a couple of weeks, the pain ceasing and the protrusion subsiding. If there was increased intraocular tension it subsided after the first suction treatment. At that time the transillumination was reported negative, but this must have been an error, as subsequently there was disclosed most unusually shaped frontal sinuses, the right being markedly clouded. This was confirmed by radiograms, which also indicated some clouding of the right antrum and that the ethmoids were clear.

In view of the X-ray findings and a persistent nasal discharge of considerable annoyance, the patient was operated upon, under local anesthesia, at Beth-El Hospital, December 5. The anterior end of the right middle turbinate was amputated, after puncturing and washing the antrum, from which the fluid returned clear. The nasofrontal duct could then be explored; it was easily entered, without resistance, with a Freeman probe at least 3 mm. in diameter.

The history of the case and the presence of polypi assured me that the ethmoid cells were altered. I therefore operated with the greatest care, using sharp biting forceps gently, with neither traction nor twisting; no force was necessary; the rasp

was used only on the anterior outer surface of the duct and with less vigor than had ever before been required. Sterile gauze packing was placed in the operated area without undue pressure. The patient was operated upon late in the afternoon and the dressing removed the next morning. Excepting that the patient fainted on the operating table, before the ethmoids were disturbed, there were no complications. She had the usual bloody discharge, and felt well for the following two days, after which she became restless and complained of backache and headache. December 16th the patient died of pneumococcal meningitis. It seems useless to give the details excepting to state that urinary examinations preceding the operation were negative. A faint trace of albumin was discovered on the third day. She never had any eye-ground or pupillary symptoms or alterations of her reflexes. Later some rigidity of the neck was noticed. She had increased leucocytosis, and a spinal puncture six days after the operation gave a cell count of 460, polys 80 per cent, with three probable pneumococci in the smear; culture negative.

DISCUSSION.

The advisability of doing intranasal operations during a more or less acute onset of the disease has always been frowned upon. This decision I have always felt wise and adhere to unless operation is forced upon me, as it was in cases 1 and 3. These cases go on record as being benefited by the procedure with no complication or dangerous sequelæ.

It has been my good fortune heretofore never to have had a fatality from any ethmoid operation. The death of this patient, unexpected, from a really simple operation, fills me with sadness and chagrin. The history of the case and the presence of polypi assured me of alterations in the ethmoid cells. It is highly probable, in view of the fact of the enormous size of the nasofrontal duct, that the frontal sinusitis was of long duration. This is confirmed by finding the polypoid condition mentioned. Care was taken when the patient was X-rayed to learn that the division between the two frontals was intact and that there were no divisions or pockets. The author is sure that there was no fault in his operative technic and believes that infection probably took place through the posterior wall of the sinus. The absence of alteration in the patient's re-

flexes made me believe that the meningitis involved the frontal areas of the brain and made my consultants feel that, at least at first, the headache was more of a uremic than of a meningeal character.

It is to be noted that in view of the severity of the orbital involvement in these cases, no intraocular involvement was seen, though after effects from retrobulbar disturbances may be found. Those that could be followed, however, have shown none.

XVI.

CLINICAL STUDIES OF THE RELATION OF THE DYSFUNCTION OF THE COCHLEAR NERVE TO THE DYSFUNCTION OF THE DUCTLESS GLANDS.*

By John Guttman, M. D.,
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INTRODUCTION.

The writer was prompted to take up the study of the relation of the cochlear nerve to the ductless glands by the examination of a patient who was sent to him for diagnosis and treatment. A study of the clinical symptoms and a close analysis of the patient's hearing power, with the usual auditory tests and the electric acumeter,[1] convinced the writer that the ear affection was caused by an endocrine disturbance. The history of the case is as follows:

N. A., 51 years old, has always been well. Family history regarding deafness negative. About 18 months ago he noticed that he could not hear the telephone well with his right ear and was compelled to use the left for that purpose. Three months later he began to suffer also from headache. He consulted a nose and ear specialist on whose advice he had an X-ray picture taken of his skull, the doctor suspecting that the sinuses were the cause of his headache. The X-ray picture showed no abnormality of his nose or ears. The physician thereupon inflated his ear a few times and declared that this was all he could do for him. As neither the headache nor the condition of his ear improved, his family physician, Dr. Brower, sent him to me for advice, stating that with the exception of an occasional low blood pressure he could find no cause for the headache, either in the circulatory, renal or alimentary tract.

Objective examination showed a physically well developed man of fairly large stature; height 5 feet 8 inches, weight 191 pounds. Pulse 72. Eyes properly corrected with concave

*Thesis accepted by the American Laryngological, Rhinological and Otological Society.

lenses. Fields of vision for white and red normal. Fundi oculi normal. Wassermann negative. Otoscopy: Drum membrane of the right, deaf, ear perfectly normal, of the left ear slightly reddened manubrium and Shrapnell's membrane. Catheterization showed slightly narrowed eustachian tubes. Caloric test showed a normal vestibular nerve. Examination of the cochlear nerve with the electric acumeter showed, as will be seen from the graph, a diminished perception of hearing of the low as well as of the high notes. Rinne on the right, the deaf ear, was negative; on the left ear, positive. Bone conduction with c_1 tuning fork, right ear slightly diminished, left ear increased. In searching for a possible cause of the headache and deafness, I asked to see the X-ray picture, which was in the possession of the patient. Studying the X-ray picture closely, I found the accessory sinuses to be perfectly normal, but the sella seemed to have a pathologic appearance. I thereupon had the same roentgenologist make an X-ray picture of the sella alone. This came back, as I had expected, with a positive finding; anteroposterior diameter, 20 mm (normal, 10 to 12 mm.); anterior and posterior clinoid processes eroded. I thereupon declared the whole symptom complex of headache, deafness on right ear, diminished hearing on left ear and diminished blood pressure to have the same cause, e. g., a dysfunction of the hypophysis gland.

This diagnosis was corroborated by the fact that the administration of the whole lobe of hypophysis extract relieved the headache. Dr. Blumgarten, who was kind enough to examine the patient endocrinologically, confirmed my diagnosis. His report was as follows:

"The patient is of the large acromegalic type with a characteristic epigastric and abdominal adiposity. He shows a distinct evidence of pituitary domination in his makeup. The headache, of course, suggests the presence of a pituitary neoplasm, but in view of the fact that he has no characteristic visual signs, I think a neoplasm can be definitely excluded."

CLINICAL EXAMINATION OF PATIENTS.

In order to investigate this much debated and quite obscure relation which may exist between the cochlear nerve and the ductless glands, the writer decided to examine patients sent to

an endocrine clinic for a possible ear affection instead of examining patients of an ear clinic for a possible endocrin affection. The endocrine clinic of Dr. Blumgarten in the Lenox Hill Hospital afforded the writer an excellent opportunity for this purpose, as the following cases were sent there for diagnosis and treatment:

1. Cases of definite endocrinopathies.
2. Cases of mental backwardness.
3. Cases of functional disturbance, without any demonstrable organic disease, which were considered by some to be due "to the domination of certain ductless glands." In relation to this Dr. Blumgarten[2] has this to say:

"Endocrinology has also fostered an important branch of medicine which we may term constitutional medicine or medical anthropology. The individual differences of people, their physiologic and anatomic variations, and their singular relations to disease, have been claimed to be due to the domination of certain ductless glands in their growth and development."

Cases manifesting such constitutional syndrome were classified under the heading "Constitutional Group."

In order to exclude the personal equation in these examinations, the histories and medical diagnoses were withheld from me until the completion of the examination of all the cases. As most of these patients were not aware of any present or previous affection of their ears, and as the writer did not know for which special symptom to look, he therefore adopted the following procedure in his examination:

1. Inspection of the Drum Membrane.—If this showed a preceding catarrhal inflammation, the case was excluded from further consideration.

2. If the otoscopic inspection showed a normal drum membrane, a functional examination of the cochlear nerve was made with loud speech and whisper across a room of about twenty feet, and with a watch of about 60 cm. normal hearing distance, with a tuning fork c_1 for Rinne and Schwabach tests (the Gelle test did not prove satisfactory to the writer). If these examinations revealed normal hearing conditions, the case was considered to possess a normally functionating cochlear nerve. If, however, the examination showed a diminished bone conduction or air conduction by watch or whisper, the

patient's hearing power was examined more minutely with the electric acumeter.

Fifty patients of the endocrine clinic were sent to me to have their ears examined; of these, the following 8 patients were excluded from further consideration:

1. Three cases for incomplete examination, having failed to appear for a second examination.

2. One case with a plus two Wassermann.

3. Four cases showing evidences of a preceding catarrhal inflammation of both ears; in three cases, where only one ear was affected, the other healthy looking ear was included in the examination. With these 8 cases deducted from the 50, there remained 42 cases for further consideration. Of these 42 patients, 22, or 52.3 per cent, showed a diminished air and bone conduction, while 5, or 11.9 per cent, showed only a diminished air conduction, without diminished bone conduction. Of these 27 cases with diminished hearing power, 22 were examined with the electric acumeter. Of this number, 15 showed a diminished perception for high or high and low notes of the tone scale, and 7 showed a diminished perception of some other part of the tone scale, as is seen from the graphs; 5 showed a diminished perception for watch and whisper only.

It is noteworthy that with the exception of three cases, none of the patients came to the clinic for ear affections, which were detected only after a minute analysis of the patient's hearing power with the electric acumeter.

As the subjective symptoms did not point to an involvement of the vestibular nerve, no examination of this nerve was made.

The fact that of the 42 patients who came to the endocrine clinic to be treated for a systemic or endocrine affection, 27, or 64.2 per cent, showed a diminished hearing power of some form was strongly suggestive that the cochlear nerve and the systemic affection had a common causative factor.

According to the clinical diagnosis made by Dr. Blumgarten, the 50 patients examined were classified in the following groups: Constitutional group, 19, or 45.2 per cent; goiter, 8, or 16 per cent; neurosis, 8, or 16 per cent; hypophysis, pregnancy, unilateral exophthalmos, undetermined mental backwardness, each 2 cases; petit mal, syphilis, abdominal syn-

drome, hay fever, menopause, each one case. No diagnosis, 3 cases.

DYSFUNCTION OF THE COCHLEAR NERVE.

In order to obtain a proper connection of the relation of the cochlear nerve to the ductless glands, it is necessary to consider the present status of these two organs.

Most cases of a nonsuppurative form of deafness are usually classified under the name of dry middle ear catarrh, otosclerosis, or progressive nerve deafness.

Bezold,[3] Politzer[4] and his school, Bruhl,[5] Alexander[6] and Frey[7] are quite emphatic in their opinion that the three above mentioned diseases are three distinct clinical entities. The symptoms which are considered to be common to all three conditions are: Negative otoscopic finding, progressive deafness, and sometimes noises in the ear. Characteristic symptoms in the differential diagnosis of typical cases are: In otosclerosis the family history usually shows a hereditary taint, the socalled Bezold triad; to this can sometimes be added a reddish reflex of the labyrinth wall, paracusis Willisii, etc. The symptoms of dry middle ear catarrh are similar to those enumerated above, and in addition there are some sequelæ of a preceding inflammatory process. Progressive nerve deafness is diagnosticated by a diminished perception of the upper tone scale, with a normal or almost normal perception for the lower tone limit. Rinne positive and diminished bone conduction.

The anatomic finding, according to the above mentioned authors, is of great importance. In making a differential diagnosis, they consider the pathologic changes in the bony labyrinth and in the oval window as characteristic of otosclerosis; changes in the middle ear as indicating dry middle ear catarrh, and changes in the membranous nervous labyrinth and the cochlear nerve as pathognomonic of progressive nerve deafness.

Manasse,[8] Hegener[9] and others, on the other hand, do not consider the three affections as distinct clinical entities, and do not believe that a differential diagnosis, especially between otosclerosis and dry middle ear catarrh, can easily be made. This opinion is also shared by most American authors. Phillips[10] in his textbook says: "Between the advanced stage of

middle ear catarrh and otosclerosis differentiation is almost impossible. Dench[11] in his textbook expresses himself in a similar fashion: "The deposit of new bony tissue in the labyrinth in the very early stages gives rise to exactly the same phenomena upon functional examination as does some obstruction to sound conduction in the middle ear." Kerrison[12] says: "When chronic catarrhal otitis media coexists with otosclerosis, the one lesion may so obscure the other that a positive diagnosis of the two conditions cannot be made."

DYSFUNCTION OF THE DUCTLESS GLANDS.

Regarding the present status of the function of the ductless glands, it may be stated that while endocrinology is a comparatively new branch of medicine, the literature on the subject is very extensive. The study of the functions of the ductless glands has contributed a great deal to the understanding of several obscure diseases, such as Addison's disease, Graves' disease, acromegaly, myxedema, cretinism, etc. However, many of its romantic and speculative contentions have not been substantiated in practice.

The ductless glands form a chain of interrelated organs; where the functions of the individual glands are sometimes overlapping, and where the hypofunction of one gland is sometimes compensated by the hyperfunction of another gland. This factor renders the diagnosis and treatment in a complicated case quite difficult. In a typical case, however, in which the symptoms of the affection of a single ductless gland are predominant, the diagnosis as well as the treatment becomes much simpler.

It was vaguely assumed long ago by some authors that some relation existed between some form of deafness, especially of the socalled otosclerosis type and a disturbance of the function of the ductless glands, because it was observed that certain forms of deafness were aggravated at puberty, childbirth, puerperium and during the climacterium.

Thyroid dysfunction, which is held as one of the etiologic factors of cretinism, was considered by Otto Mayer,[13] Bloch,[14] Alexander[15] and others, also to be responsible for the accompanying deafness or deafmutism in some cretins. It is difficult to state what relation exists between the thyroid gland, the

hypophysis and the other ductless glands, as this question is still *sub judice;* but so far as the ear is concerned, the dysfunction of the hypophysis seems to play an equally, or possibly a more important part than the dysfunction of the thyroid gland. According to Kessel and Hyman,[16] "the role of the thyroid in the production of clinical symptoms is greatly overemphasized."

The case of N. A., cited above, illustrates in a striking manner, the relation of the hypophysis to cochlear nerve affection.

Denker,[17] in a paper read before the Boston Otological Congress in 1912, also attempted to show a relation existing between the dysfunction of the hypophysis and otosclerosis, which he considers a definite clinical entity.

The presence of a dysfunctionating pharyngeal hypophysis in the proximity of Luschka's tonsil may be the underlying cause of the injurious effect upon the ear which is usually attributed to the adenoids and tonsils; adenoidectomy, therefore, removes not only the mechanical pressure of the adenoids upon the eustachian tube, but may possibly also bring about a change in the function of the hypophysis. This may explain the beneficial effect of adenoidectomy upon some ear affections.

Citelli[18] attempted to connect the improved growth of a child after adenoidectomy with the contemporary removal of the pharyngeal hypophysis, inasmuch as the hypophysis is supposed to influence the bony growth. Bryant[19] also tried to influence the pharyngeal hypophysis in ear affections by applying nitrate of silver to the pharynx.

SUMMARY.

The following facts seem to point towards a possible relation between the cochlear dysfunction and endocrin disturbance:

1. The case of N. A., detailed above, where deafness was associated with a dysfunction of the hypophysis.

2. That out of 50 patients suffering chiefly from an endocrine or other systemic affection, a high percentage (64.2 per cent) were found to be suffering also from some form of diminished hearing power. In this connection the charts obtained by the use of the electric acumeter have great illustrative value.

3. The fact that certain forms of deafness often become ag-

gravated during puberty, gravidity, puerperium, climacterium, etc., where the ductless glands seem to play an important role.

4. Deafness is often encountered in cretinism, which is also the result of an endocrine disturbance.

5. Improved hearing following adenectomy may be the result of the removal of the pharyngeal hypophysis.

6. The above named three types of ear affections, as dry middle ear catarrh, otosclerosis and progressive nerve deafness, may in a great number of cases be considered as one, and if clinical and laboratory examinations point towards some endocrine involvement, they may be designated as idiopathic deafness of endocrine origin.

7. In the functional examination of the ear, if no other constitutional cause can be found, diminished bone conduction is suggestive of idiopathic deafness of endocrine origin. According to the figures above, 52 per cent of the patients frequenting an endocrine clinic, and referred for ear examination, showed diminished bone conduction.

Rinne negative and prolonged bone conduction may mean only an advanced stage of the disease where stapes ankylosis has already set in.

8. Idiopathic deafness of endocrin origin may be considered as an affection of the sound perceiving apparatus, and may be differentiated from progressive nerve deafness by the fact that its etiologic cause is some endocrin dysfunction. In many of these cases there was a diminished perception not only of the high but of the low notes of the tone scale also, as the appended graphs clearly show.

9. In idiopathic deafness of endocrin origin, as in many endocrinopathies and nerve disorders, heredity is an important factor.

10. The treatment, as in other nerve affections, must, for the present, consist in constitutional treatment, rest, proper surroundings, proper diet, etc. In the early stages some medical or endocrin treatment may be of some value.

11. Even though the patient does not complain of any ear affection, the endocrinologist may utilize the functional examination of the cochlear nerve for differential diagnostic purposes similarly to the manner in which he utilizes the changes in the field of vision or the optic nerve.

While the number of patients examined clinically by the writer is rather small (50), and while there are no anatomic findings to corroborate the clinical findings, still the results of these functional ear examinations seem to warrant, at least tentatively, the conclusions set forth above.

Series	Name	Age	Gender	OTOLOGIC DIAGNOSIS								MEDICAL DIAGNOSIS							REMARKS
				Dim. bone cond'n (Schwabach)	Dim. perc'n of high or high & low notes	Dim. perc. of other part of tone scale	Dim. perc., watch and whisper only	Catarrhal	Incomplete	Normal	Constitutional group	Goiter	Hypophysis	Neurosis	Pregnancy	Menopause	Incomplete	
1.	M. B.	16	F.	+	+	....	....	....	....	....	+	....	....	....	....	....	....	
2.	O. J.	52	....	+	+	....	....	....	....	....	+	....	....	....	....	....	....	
3.	F. L.	35	....	+	+	....	....	....	....	....	....	....	....	....	....	....	....	
4.	A. N.	51	M.	+	+	....	....	....	....	....	....	....	+	....	....	....	....	
5.	M. T.	19	....	+	+	....	....	....	....	....	....	....	....	....	....	....	....	
6.	C. M.	59	F.	+	+	....	....	....	....	....	....	....	....	....	....	....	....	Petit mal
7.	S. S.	52	....	....	....	....	....	....	....	....	+	....	....	....	....	....	....	
8.	C. A.	54	....	....	....	....	....	....	....	....	....	....	....	....	....	+	....	A.S. Sequelæ O.M.P.A.
9.	E. F.	52	....	....	....	....	....	....	....	....	....	....	....	+	....	....	....	R.
10.	B. H.	42	....	+	+	....	....	....	....	....	....	+	....	....	....	....	....	A.S. Sequelæ O.M.P.A.
11.	B. A.	38	....	+	+	....	....	....	....	....	+	....	....	....	....	....	....	
12.	G. S.	44	M.	+	+	....	....	....	....	....	....	....	....	....	....	....	....	
13.	A.	23	....	+	+	....	....	....	....	....	+	....	....	....	....	....	....	
14.	O.	69	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	Multiple sclerosis
15.	S. B.	53	F.	....	....	....	....	....	....	....	....	....	....	....	....	....	....	AD. Mastoid operation
16.	M. T.	34	....	+	....	+	....	....	....	....	+	....	....	....	....	....	....	
17.	N. F.	25	....	+	....	+	....	....	....	....	+	....	....	....	....	....	....	
18.	R. A.	22	....	+	....	+	....	....	....	....	+	....	....	....	....	....	....	
19.	H. K.	34	....	+	....	+	....	....	....	....	+	....	....	....	....	....	....	
20.	F.	32	....	+	....	+	....	....	....	....	....	....	....	+	....	....	....	
21.	K. E.	34	....	+	....	+	....	....	....	....	....	....	....	+	....	....	....	
22.	K. R.	14	....	+	....	....	....	+	....	....	+	....	....	....	....	....	....	
23.	W. K.	26	....	+	....	....	+	....	....	....	....	+	....	....	....	....	....	
24.	P. T.	12	M.	+	....	+	....	....	....	....	....	+	....	....	....	....	....	
25.	W. C.	54	F.	+	....	....	+	....	....	....	....	....	....	....	....	....	....	Unilateral Exophthalmos
26.	R. E.	38	F.	+	....	....	+	....	....	....	....	+	....	....	....	....	1	

				OTOLOGIC DIAGNOSIS						MEDICAL DIAGNOSIS									
Series	Name	Age	Gender	Dim. bone cond'n (Schwabach)	Dim. perc'n of high or high & low notes	Dim. perc. of other part of tone scale	Dim., perc., watch and whisper only	Catarrhal	Incomplete	Normal	Constitutional group	Goiter	Hypophysis	Neurosis	Pregnancy	Menopause	Incomplete	REMARKS
27.	K. F.	15	F.	....	....	....	+	....	....	....	....	....	....	+	....	....	....	
28.	L.	42	F.	++	....	....	....	....	....	....	....	+	....	....	....	....	....	Wassermann
29.	L. M.	34	F.	....	....	....	....	++	....	....	....	....	+	....	....	....	....	
30.	R. P.	37	F.	....	....	....	....	++	....	....	....	....	....	....	....	....	....	
31.	W. E.	42	F.	....	....	....	....	++	....	....	+	....	....	+	....	....	....	
32.	S. T.	34	F.	....	....	....	....	....	....	....	....	....	....	....	....	....	....	
33.	D. G.	39	F.	....	....	....	....	....	++	....	+	....	....	....	....	....	....	Undescended testis; abdominal syndrome
34.	B. A.	11	M.	....	....	....	....	....	....	....	....	....	....	....	....	....	....	
35.	W. A.	39	F.	....	....	....	....	....	+	....	+	....	....	....	....	....	....	
36.	P. E.	32	F.	....	....	....	....	....	....	+++	....	+	....	....	....	....	....	
37.	K. A.	19	F.	....	....	....	....	....	....	+++	....	....	....	....	....	....	....	Unilateral Exophthalmos
38.	K. F.	43	F.	....	....	....	....	....	....	....	....	....	....	....	....	....	....	
39.	L. G.	34	M.	....	....	....	....	....	....	+	+	....	....	....	....	....	....	
40.	M. G.	26	M.	....	....	....	....	....	....	+++	+	....	....	....	....	....	....	
41.	W. L.	18	F.	....	....	....	....	....	....	+++	+	....	....	....	....	....	....	
42.	S. A.	19	F.	....	....	....	....	....	....	+++	+	....	....	....	....	....	....	
43.	S. F.	14	F.	....	....	....	....	....	....	+++	+	....	....	....	....	....	....	
44.	Z. L.	31	F.	....	....	....	....	....	....	+++	....	+	....	....	....	....	....	
45.	R. A.	32	F.	....	....	....	....	....	....	+++	....	+	....	....	....	....	....	
46.	L.	16	F.	....	....	....	....	....	....	+++	....	....	....	+	....	....	....	
47.	B. R.	30	F.	....	....	....	....	....	....	+++	....	....	....	+	....	....	....	Hayfever
48.	P. O.	32	F.	....	....	....	....	....	....	+++	....	....	....	....	....	....	....	Undetermined backwardness
49.	G. M.	10	F.	....	....	....	....	....	....	+	....	....	....	....	....	....	....	Undetermined backwardness
50.	G. W.	8	F.	....	....	....	....	....	....	....	....	....	....	....	....	....	....	

MEDICAL EXAMINATION.

Constitutional Group	Dim. bone conduction (Schwabach)	Normal bone conduction	Excluded	Dim. perception of high or high and low notes	Dim. perception of some other part of tone scale	Dim. perception of watch and whisper	Catarrhal	Incomplete	Wassermann ++	Normal
19	9 47.3%	7	3	7 36.8%	4 21%	..	1	2	..	5 26.3%
Goiter 8	2 25%	5	1	1 12.5%	1 12.5%	2 25%	1	..	..	3 37.5%
Neurosis 8	4 50%	3	1	2 25%	2 25%	1 12.5%	12.5%	..	..	2 25%
Scattered 15	46%	5	3	33.3%	..	2	1	1	1	5

OTOLOGIC EXAMINATION.

Patients examined, 50

Female, 41		Male, 9	
Complete otologic examination	Incomplete	Specific	Catarrhal
42	3	1	4

Forty-two complete otologic examinations

Diminished bone conduction (Schwabach)	Normal bone conduction
22, or 52.2 per cent	20, or 47.8 per cent

Electric acumeter examination	Diminished perception for watch or whisper	Normal
22	5	15

Twenty-two electric acumeter examinations

Diminished perception for high or high and low notes of tone scale.	Diminished perception of some other part of tone scale.
15	7

Conventional signs used in plates: V, conversational voice; v, whispered voice; h, watch; W, Weber; R, Rinne; mt, membrana tympani; Au, both ears; AD, right ear; AS, left ear; Ac, ad concham; 20′ X—more than 20 feet; M, male; F, female. Fraction following V and v designates feet. Fractions following h designates centimeters. The numerator of the fraction indicates the right ear. The denominator of the fraction indicates the left ear.

In the curve the solid line represents the hearing power of the right ear, the dashed line the hearing power of the left ear, and the dotted line the normal hearing power.

Where on account of a great diminution of hearing power the ordinary intensity of sound is not sufficient, a sound of greater intensity, which is about four times greater than the normal, by eliminating some resistance in the apparatus, is produced.

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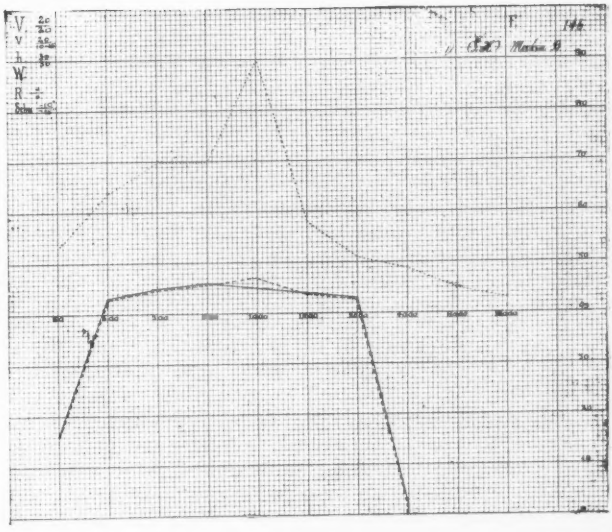

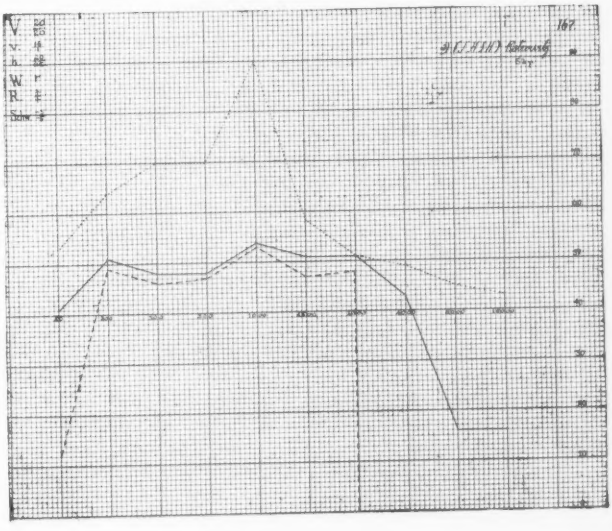

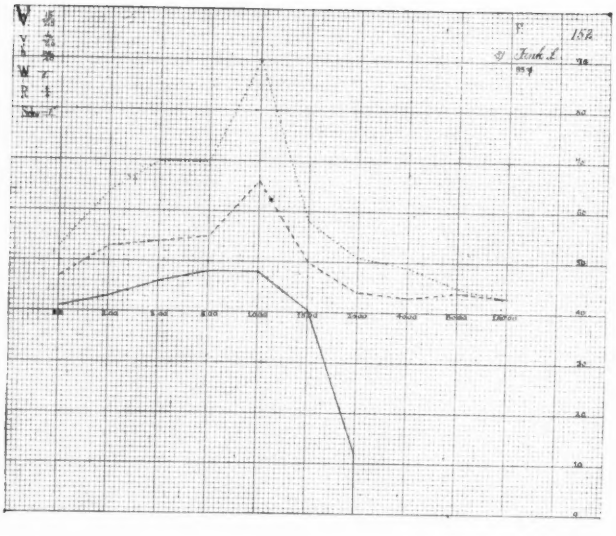

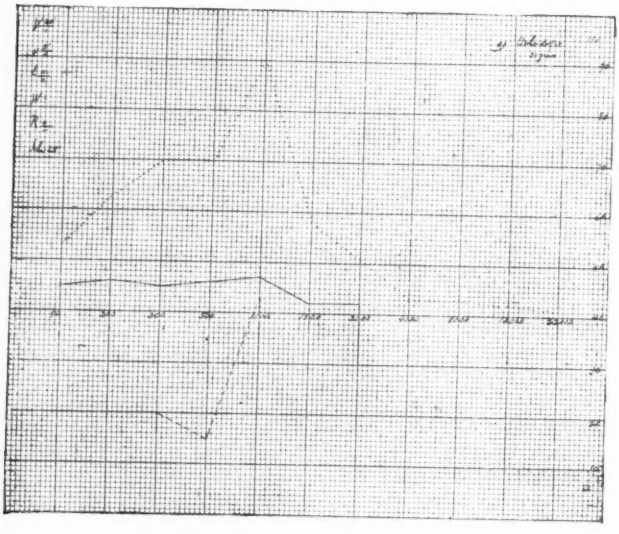

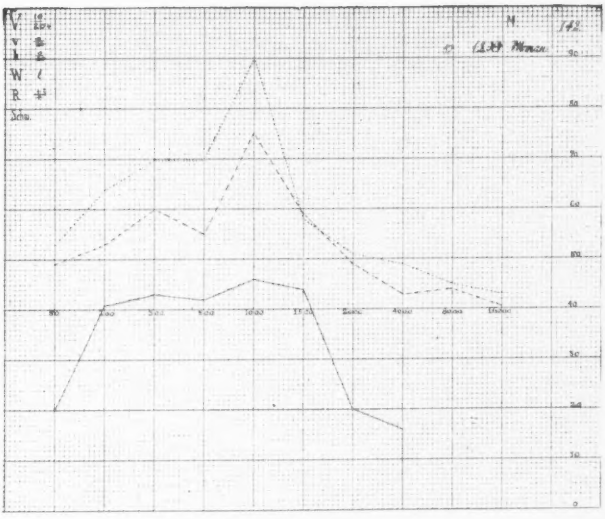

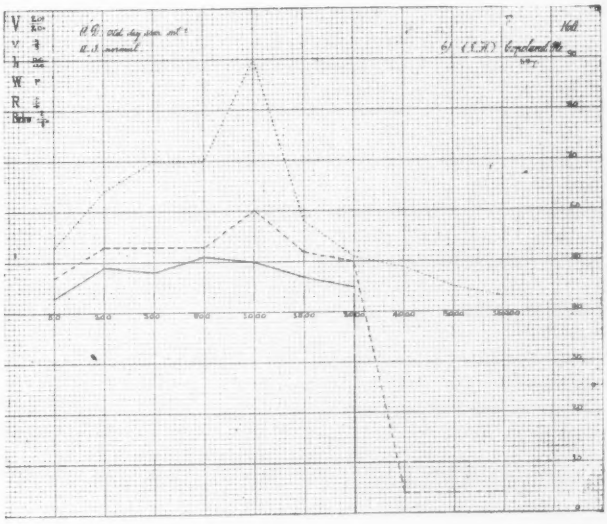

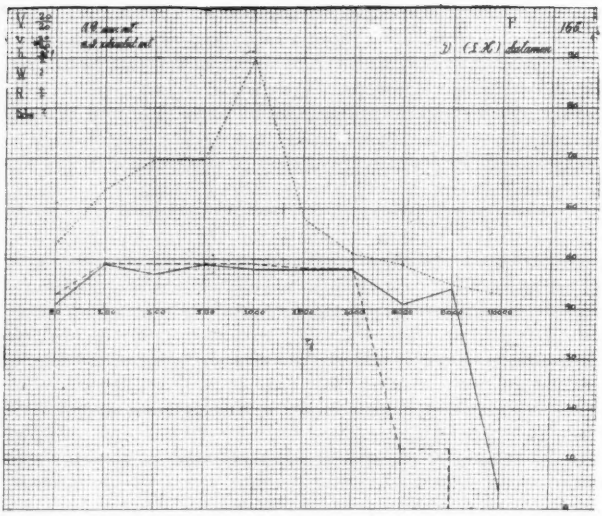

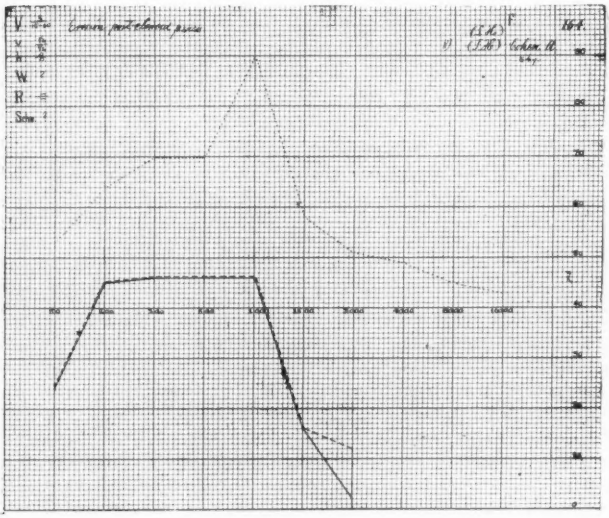

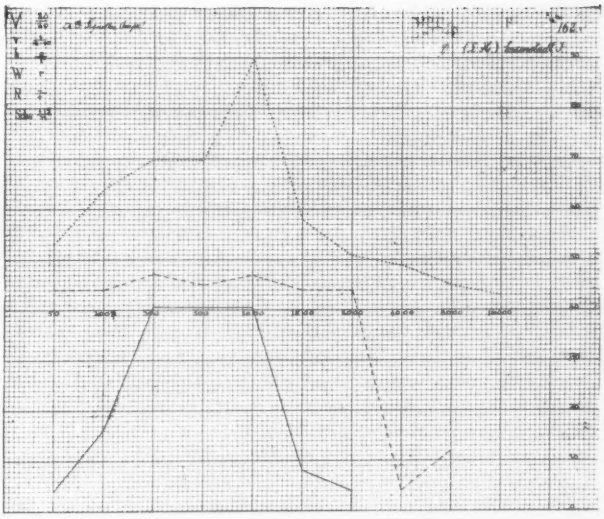

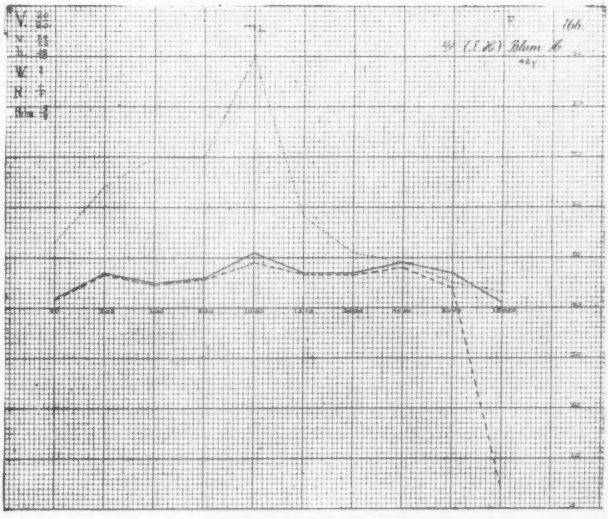

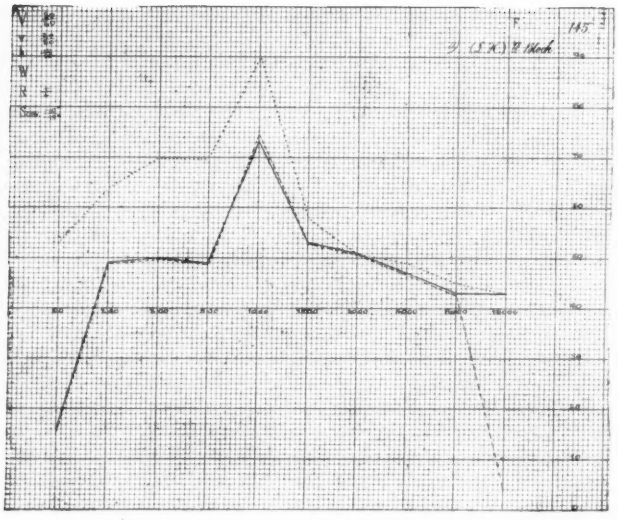

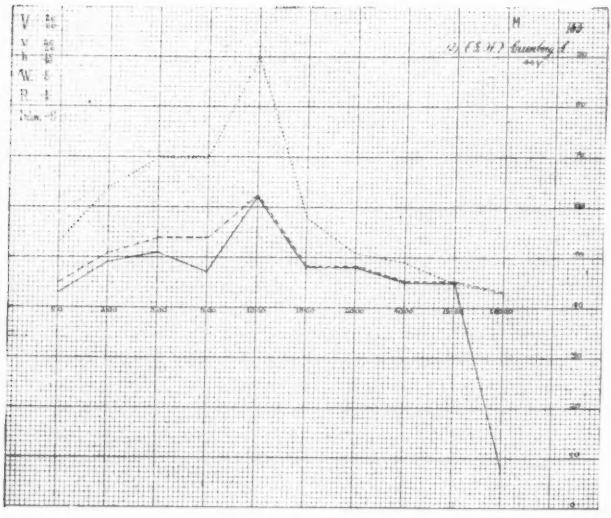

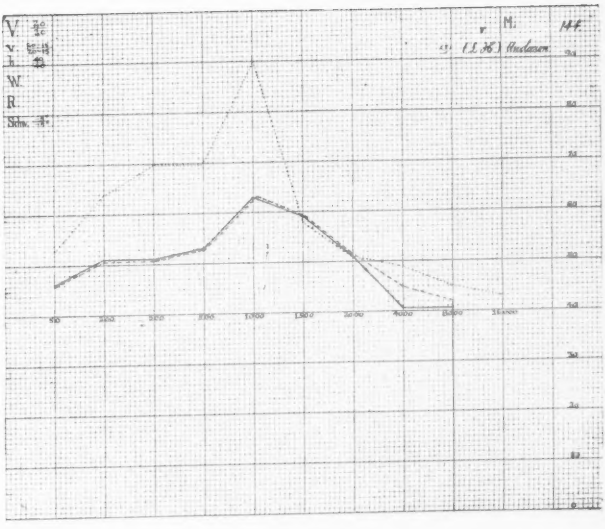
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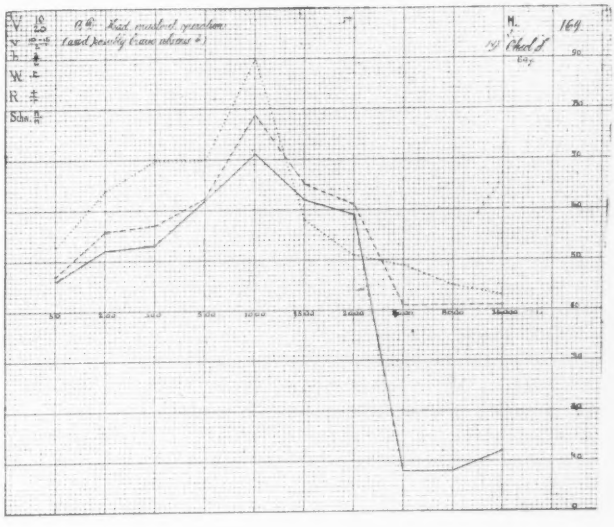
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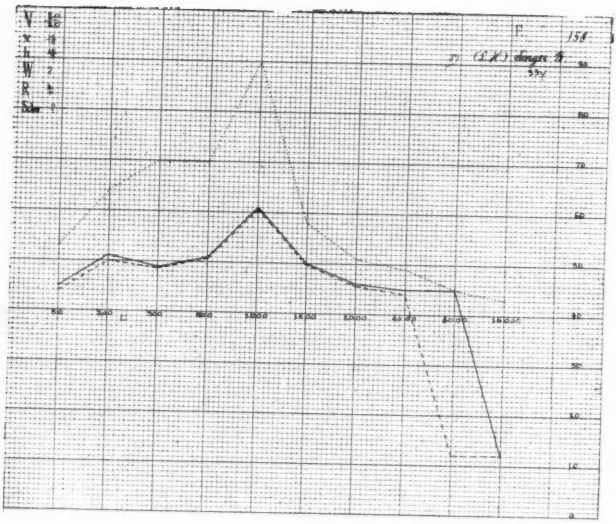

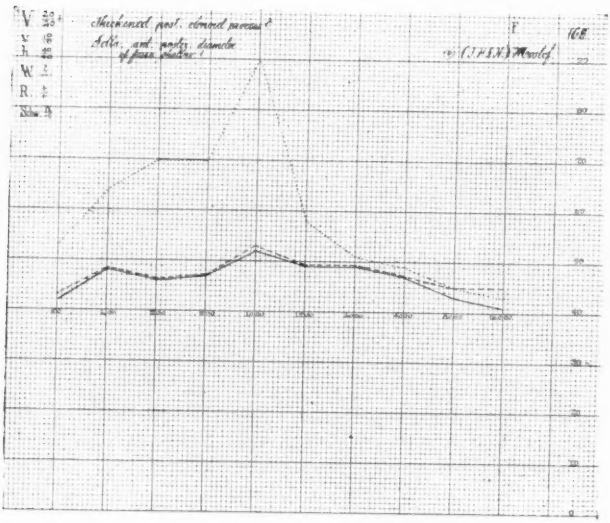
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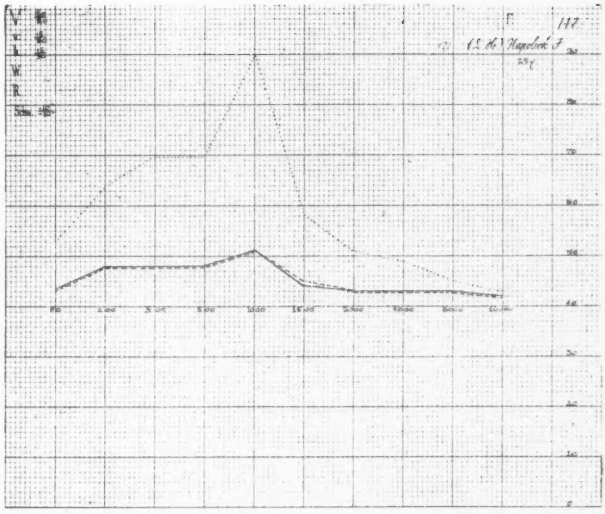

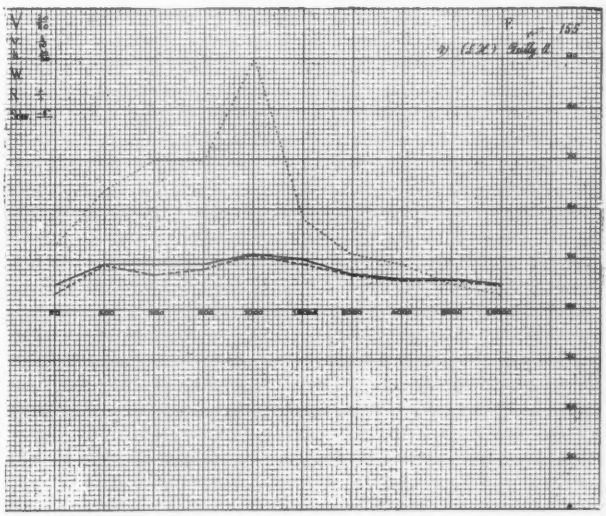

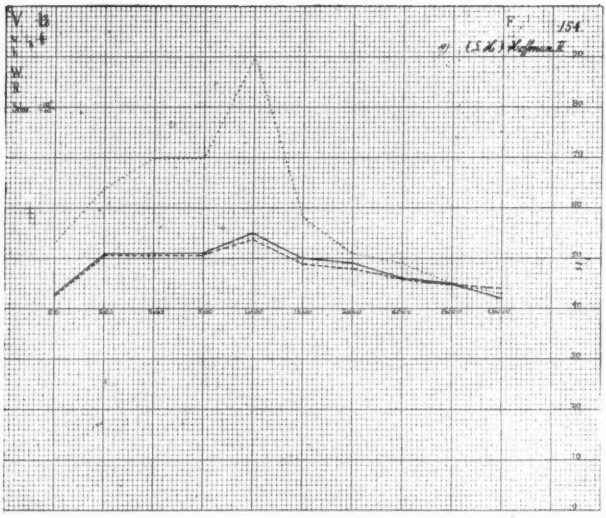
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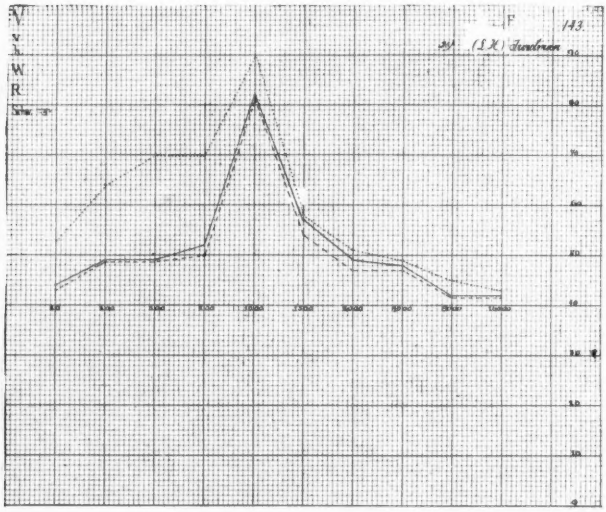
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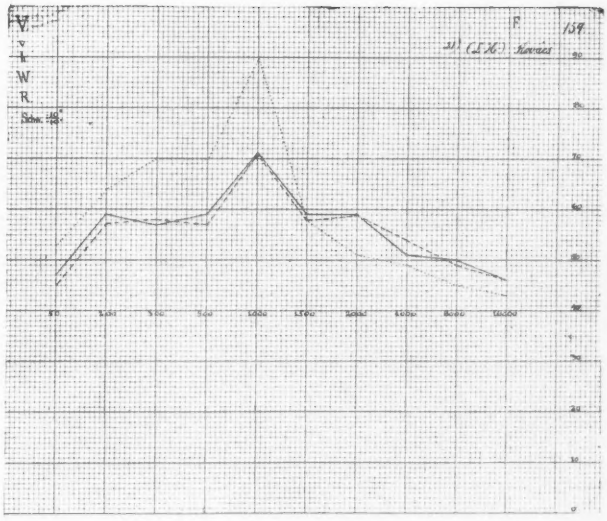
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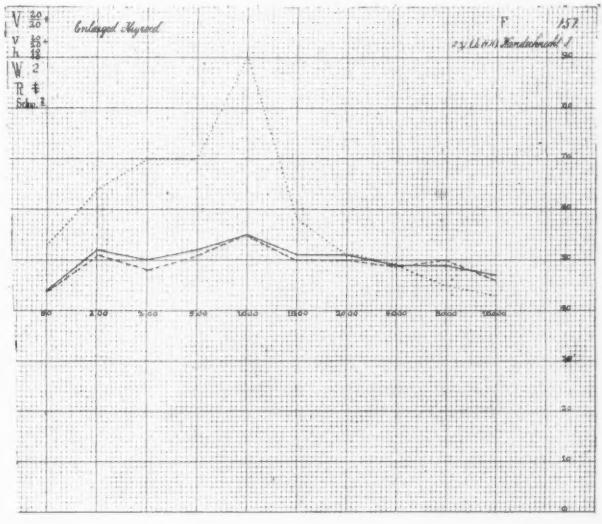
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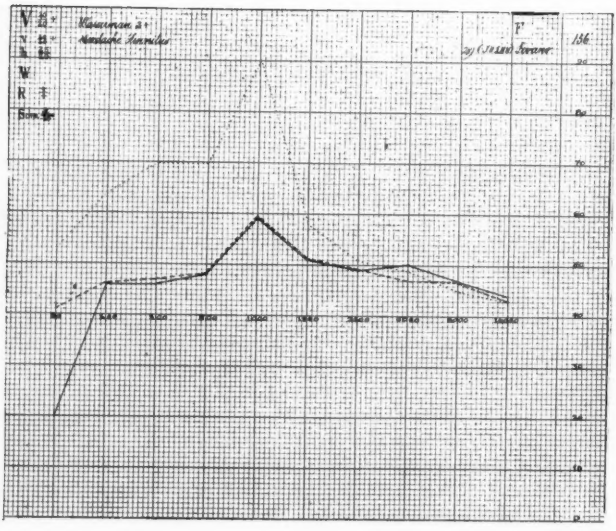

XVII.

EMPYEMA OF THE ANTRUM OF HIGHMORE OF SCARLATINAL ORIGIN.*

By Virginius Dabney, M. D.,

Washington, D. C.

It seems moderately certain that scarlet fever owes both its origin and its contagious nature to the presence of streptococci of especial virulence in the secretions of the nose and throat of individuals who have been rendered susceptible to particular strains of these organisms. The disease is thus transferred to others by actual droplets or exhalations from the nose and throat containing these streptococci, whose responsibility in the causation of scarlet fever has been still further established by Doctors George F. and Gladys H. Dick, who have experimentally produced the disease, using a filtered serum of streptococci. Their experiments are conclusive and highly valuable as they conform strictly to Koch's dicta and contribute so substantially to the study of this justly dreaded disease that they offer hope for the creation of artificial immunity to it. Caronia and Sidoni of Rome and Dochez have published confirmatory reports of this nature. The frequent involvement of the middle ear and mastoid process in cases of scarlet fever is, of course, familiar and for the foregoing reasons easily understood, yet the alleged escape of the nasal accessory sinuses in this involvement is, I believe, more apparent than real. There is nearly always a marked hyperemia of the nasal mucosa with the inevitable stimulation of the mucous glands and outpouring of thick mucus, but the actual blocking of the nares and sinuses, which I consider not rare, has escaped our notice in the presence of more urgent and distressing symptoms, only too common in this disease. Such is my mature belief after making a special study of this phase of scarlatina in the isolation pavilion of Garfield Hospital for

*Read before the American Laryngological Association, June 3, 1924.

three years. In fact, my attention was forcibly drawn to this complication by my attempts to relieve the acute distress of a colleague suffering from scarlet fever in whom the nasal and throat symptoms were especially distressing. I found that great relief was afforded him by frequent irrigation with hot saline solution of the throat and nose, especially the latter, really immense quantities of thick secretion being brought away at each irrigation. Wherever the slightest evidence of sinus involvement exists I make this treatment a matter of routine, when I have been brought into a case. With very young children it is, of course, frequently impossible and even dangerous, but I have generally been able to accomplish my wishes in this respect by the exercise of gentleness and tact: indeed, they come to look forward to it with pleasure, so great is the relief. At least, it is well worth the time to try to get them to agree to it, as I am sure I have decreased markedly the percentage of ear involvement thereby. Unfortunately I see these cases generally only when the ear complication has already been established, along with a sinus involvement, but occasionally when called in to give an opinion on the ear and throat condition, I am able to institute the nasal irrigation. The mouth breathing and excoriation of the nasolabial margin, so common in these cases, in themselves suggest the necessity of some measure for the relief of the basic condition—nasal infection and blocking. Thus, assuming my premises as correct, it would appear that in a majority of cases the sinuses do share in the streptococcic infection of the nose and throat, and that the latter is an invariable condition in scarlatina. However, despite my belief that sinus involvement is fairly common here, I have seen but one case of true empyema directly due to scarlatina, and it is with this case that this paper is mainly concerned.

The patient, a vigorous, well nourished pupil nurse, had been confined to bed for several days, after a sudden onset, with a tentative diagnosis of influenza, when I saw her in response to a request to investigate an apparent blocking of the nose and slight pain in the ears. Her symptoms were rather vague, being merely a temperature of 99.2 to 100, albuminuria, hyaline casts, headache (chiefly frontal), backache, stoppage of the nose and slight pain in the ears. Later,

her temperature had a wider excursion, going to 102, in the evening. Both drums were normal, save a slight retraction, and there was slight loss of hearing for the conversation voice, doubtless due to this and the partial closure of the tube. The throat was red, perhaps a little uncomfortable on swallowing (due to mouth breathing, probably), and the normally atrophied tonsils apparently innocent and free of secretion. The patient herself laid great stress on the feeling of weight and pressure in the face, and seemed to think that all her trouble lay there, certainly all her discomfort. The nose proved to be greatly engorged, the turbinates being much swollen and red, free mucopurulent secretion was seen, but irrigation was only partly successful in ridding the nose of this accumulation, as the intumescence of the turbinates subsided but little even under cocain and adrenalin. Cultures from both nose and throat showed hemolytic streptococcus in nearly pure culture. While she complained of a sense of fullness in one side of the face, there was no localization over the antrum; in fact, pressure anywhere on the brow, cheek, side of the nose and malar bone gave pain. As she was not my patient primarily I saw her only from time to time, but her history for the first two weeks was substantially as just given. She was getting at the hands of a competent nurse irrigation of the nose two or three times a day, which always brought away a great deal of tenacious mucus, sometimes purulent. When her active symptoms somewhat abated she decided to go to her home in the city and I saw her three weeks later, that is, after six weeks' illness. At this time her condition was serious; her skin was yellow, her feelings close to melancholia and her general appearance that of the chronic nephritic. She had an evening temperature of 100, arthritis of the knees, ankles and fingers, and, the next day I got a report on her urine and blood showing a heavy trace of albumin and 42 per cent hemoglobin. Bright yellow pus was welling out of the infundibulum and transillumination was definitely confirmatory of antrum infection. At this time there was localization of all her discomfort over the antrum area and nowhere else. After daily washing out of the antrum and the administration of Fowler's solution of arsenic, her hemoglobin rose to 55 per cent and I considered it safe to do a Caldwell-Luc operation, which

disclosed a cavity full of pus under pressure and some polypoid degeneration of the mucosa. Cultures of this pus showed the hemolytic streptococcus consistently throughout her convalescence, and it was the persistence of this organism and its sustained morphologic behavior that excited my interest and suspicion of scarlet fever. I had recently seen so many cases in consultation of anomalous forms, all with this same organism apparently playing an important part, that it occurred to me possible that here was a case of scarlet fever manifested as a local infection. Thus, I traced her case from the very beginning and found that she had come down with her illness four days after she had left two cases of scarlet fever. Moreover, she had never had the disease herself and her medical attendant told me that he was convinced she then had it, despite the absence of the ordinary symptoms. Thus, her original illness was not influenza complicated by an antrum infection, but was scarlet fever exhibited as an infection of the antrum, a vicarious scarlatina, as it were. The organism repeatedly obtained from the antral secretion was morphologically identical with those obtained from the noses and throats of the cases with which she had had contact, a fact I happened to be able to establish purely by chance. I had been trying to connect these cases with a mild local epidemic of complications associated with scarlatina, chiefly mastoiditis, in the isolation pavilion and thus happened to have all the cultural evidence at hand. If scarlatina is merely an anaphylaxis to the streptococcus in certain virulent forms and often shows itself only in the throat, why may we not say that this patient had scarlatina with its manifestation only in the antrum? Further corroboration exists in the fact that her roommate developed scarlatina a few days after my patient left the dormitory.

An unusual incident of the convalescence nearly ruined all the benefit of the operation. One morning I noticed that the wick I kept in the canine fossa wound was missing, and, upon questioning, she said that at dinner the night before she had swallowed it; as it was only two inches long, this could readily have happened, and I naturally accepted her statement. At first she did very well but after three weeks she seemed to retrograde and complained constantly of a

feeling of weight in the cheek. Moreover, the pus continued in volume and odor, and an autogenous vaccine gave a violent reaction but no general benefit. Her anemia likewise continued despite intramuscular injections of iron. Daily irrigation of the antrum apparently did no good and I began to be much discouraged till one afternoon, after she had already had her morning treatment, during which I had probed the antral cavity laboriously and used considerable force in the irrigation, she returned to say that the gauze had fallen into the back of her mouth a few moments before and she had spit it out. Evidently the gauze had fallen into the antrum and not been swallowed, and had lain there for nearly three weeks, finally to be pushed or washed into the open naris and thence into the postnasal space and mouth. In three days the entire clinical picture had changed; no pus was seen, the weight in the cheek was gone, the evening temperature became normal and her entire outlook on life was natural. The anemia and albuminuria, however, required an additional three weeks to disappear.

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XVIII.

OTOLOGIC OBSERVATIONS—OCULAR RESPONSES TO VESTIBULAR STIMULATION.

By E. M. Josephson, M. D.,

New York.

The nature of the vestibular phenomena is still an unsolved problem. Inasmuch as any additional data are of value in casting light on this problem, the writer does not hesitate to present the following observations and data of incompleted experimental work upon the vestibular reactions.

Study of the relation of vestibular stimulation to the ocular reactions has revealed that there is in the normal individual a variation in the duration of nystagmus with the variation in the length of the period of vestibular stimulation, and also a definite relation between the rate of stimulation and the rate of the nystagmus movements. The mode of stimulation adopted in the experiments was rotation, because it more nearly resembled the normal form of stimulation of the vestibular apparatus than any other form at our command. The rate of rotation was determined in terms of time required for ten rotations, and the rate of nystagmus, in terms of the time required for ten nystagmus cycles. The results are noted below:

Case 1.—A. L., age 10 years. Vestibular apparatus was normal.

Case 2.—D. P., age 22 years. Vestibular apparatus was normal.

Case 3.—S. M., age 24 years. History of left chronic otitis media, necessitating two mastoid operations. There were symptoms of vestibular irritation in the history (attacks of headache and vertigo and proneness to seasickness recently set in), and also in the physical examination (diminution in the period of nystagmus on rotation toward the side.

Case 1.—Seconds required for 10 rotations	10	15	20	25	30
Seconds' duration of 10 nystagmus cycles	7	10	14	15	No nys.
Case 2.—Seconds required for 10 rotations	12	15	20	30	
Seconds' duration of 10 nystagmus cycles	3	5	14	20	

In these cases the initial nystagmus movements were noted to be more rapid than the subsequent.

	On Rot'n to Rt.			On Rot'n to Lt.	
Case 3.—Seconds required for 10 rotations	10	20	12	20	22
Seconds' duration of 10 nystagmus cycles	5	10	5	5	5

In this as well as in other cases presenting signs of vestibular irritation, the response of the diseased vestibule was maximal, whether the stimulation be minimal or maximal.

Rotation and other of the generally used methods of maximal stimulation of the vestibular apparatus cannot be regarded as representing the normal mode of vestibular stimulation and, inasmuch as those types of stimulation are maximal, the responses must be regarded as in part the manifestations of overflow of the stimulus to adjacent or related portions of the nervous system. A priori, it is obvious that a mechanism serving the function which the vestibular is regarded to serve must respond to normal and minimal forms of stimulation. A study was made with an idea to determine what, in addition to the eye rolling movements, might be the form of ocular response to minimal vestibular stimulation. The mode of stimulation adapted was one, two or three rotations, the number chosen being always below that necessary to create nystagmus. The normal cases presented after stimulation a heterophoria, and the cases which previous to rotation had shown a muscle imbalance thereafter showed an alteration of heterophoric reactions.

The third observation has recently been described by the writer under the designation of the oculovestibular past point-

ing reflex. It is elicited as follows: The patient, whose vestibular apparatus has not been previously stimulated, is asked to point with eyes open and then with eyes closed. With eyes still closed, the patient is then asked to rotate his eyes in the direction of the arm which has pointed, and to point once again. In the majority of normal cases the patient will past point in the direction toward which the eyes have been rotated. The past pointing reaction is absent in patients presenting labyrinthine lesions or a dead labyrinth, on the side of the lesion, but can be elicited on rotation of the eye toward the side of the normal vestibular apparatus, and pointing with the corresponding arm. Contrary to the opinion of Barany, past pointing can seldom be elicited in normal individuals by a rotation of the head. On the other hand, if the head be first rotated and then the eyes still further rotated in the same direction, the past pointing may be elicited.

A fourth observation, which has recently been confirmed in the findings of other observers, including Dr. Pearce, appears to be in the nature of a "spread" reaction. Cases presenting irritative lesions of the facial nerve of otic etiology manifest upon stimulation of the vestibule involved a nystagmoid flicker movement of the upper lid of short duration. Similar reactions have been reported by experimental observers of various lower vertebrates.

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230 E. Seventy-ninth St.

XIX.

RADICAL MASTOID OPERATION FOLLOWED BY PARESIS OF THE SEVENTH, FIFTH, AND A PORTION OF THE THIRD CRANIAL NERVES —RECOVERY. ADVANTAGES OF THE BARANY MODIFICATION.

By H. W. Lyman, M. D., F. A. C. S.,

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St. Louis.

The patient was a young woman, eighteen years of age, who had always been healthy except for the usual childhood diseases, and whose family history was negative, except that five brothers and one sister died in infancy. There was no history of cancer, tuberculosis, lues, or nervous disorders.

About ten years ago this patient developed a right acute otitis media a few hours after swimming. She was treated at different times by various physicians. During this period there were frequent recurrent attacks, the discharge lasting three or four months. There was one quiet period of one year and another of three years. Pain of variable intensity was present during the exacerbations. The present one began about six months previous to her admission to the hospital. She has had frequent attacks of headache in right parietal and frontal regions.

The physical examination was negative except as to the right ear. The pupils were equal, circular, and reacted to light and accommodation. The external ocular muscles were normal. There was no conjunctivitis. The reflexes, ankle jerks, knee kicks, biceps and triceps were equal and active on both sides. There was no Babinski. The Wassermann reaction was negative; the temperature was 98.8; and the leucocyte count was 10,000.

The right ear showed a moderate purulent discharge with a foul odor and a polypoid mass protruding through a perfo-

ration in the posterior inferior quadrant. There was some tenderness in the region of the mastoid antrum. The patient heard the whispered voice at about six feet. The deafness was of the conducting mechanism type. The left ear was normal.

The X-ray examination showed an entire absence of any cellular structure in the right mastoid. The left mastoid was normal.

On October 14, 1922, Dr. Robert Barany demonstrated his modification of the radical mastoid operation on this patient. The incision was made close to the auricle. The bone of the mastoid process was chiseled away in the usual manner, removing the posterior bony canal wall. Great care was taken not to disturb the membranous canal, the object of this operation being to preserve that structure intact. The antrum was found deeply seated. It was very small and filled with cholesteatomatous material. The "bridge" and the outer wall of the attic were next removed, also without disturbing the membranous canal. The incus was then removed, and as cholesteatomatous material was seen anterior to the malleus, the latter was also extracted, bringing with it most of the drumhead. The bony cavity was cleaned out thoroughly. The bone was necrotic around the tunnel containing the tensor tympani muscle. This region was curetted very thoroughly. The dura was slightly exposed above and behind the site of the antrum, but it appeared to be in normal condition. The incision was closed completely, except at the lower end, where a small rubber tissue drain was inserted. The ear canal was lightly tamponed with cotton and the usual dressing applied. At the termination of the operation the corneal reflex was present, showing that there was no involvement of the seventh nerve. The patient was returned from the operating room in good condition except for a feeling of dizziness.

On October 15th the patient was still dizzy. Her temperature was normal.

On October 16th her general condition was good, and the dizziness had disappeared.

While the first dressing was being done, the rubber tissue drain was torn during removal and apparently about one-third remained in the wound. One of the skin clips was loosened

and the retained piece of rubber tissue extracted. A rubber glove drain was inserted and the metal clip replaced. The removal of the cotton tampon from the canal was very painful, and the canal seemed unusually sensitive.

Early in the morning of October 17th (sixty hours after the operation), the patient noticed an irritation of the right eye with continuous lacrimation. Examination showed no obvious cause for this condition. In the afternoon (seventy-two hours after the operation), a laxness of the right side of the face was noticed and the patient complained of a peculiar sensation on that side. A rough, sensory test at this time showed a diminution of pain perception on the right side of the face over the area supplied by the third branch of the fifth nerve. Light touch was also slightly impaired over the same area. Deep pressure was not involved. Right corneal anesthesia was present. Pupillary reflexes were normal. At the same time it was noted that the right eyelid could not be closed. That evening the patient complained of numbness of the right side of the face. There was practical insensibility to pain and light touch over entire area supplied by right trigeminus. The trigeminal motor function was difficult to determine. No weakness of the masseter or temporal muscles could be made out on either side by palpation. When the mouth was opened there was a suggestion of a deviation of the jaw to the left, and the patient said she could not abduct the jaw to the right.

On October 19th the facial paresis (seventh nerve) was complete. The patient complained of pain in the right side of the face, which was still insensible to touch and pin prick. Sensations of sweet, bitter, acid, and salt were unimpaired on the tip of the tongue. Patient was able to open her mouth much wider today than yesterday and with very slight deviation to the left.

On October 20th the neurologic consultant reported that there was no trigeminal hypesthesia or other sensory defect in the trigeminal region at this examination, except some difference in the corneal and conjunctival reflexes of the two eyes, the left being very much more active. The pupils were widely dilated. The possibility of an intracranial complication was suggested.

On October 21 the ophthalmologic consultant reported that there was a paresis of the levator palpebrae superioris and the superior rectus of the right eye, both of which are supplied by the same branch of the motor oculi nerve. The eyegrounds were normal. The pupils were equal and reacted actively to light and accommodation.

On October 22nd, the patient had diplopia on looking upward, and on reading, the image of the right eye was blurred.

On October 25, 1922, the patient had no apparent paresis of the levator palpebrae superioris but still had double vision on looking upwards.

On October 26th the patient complained of pain in the right ear during the previous night. The ear (which had been dry) had been discharging for the last thirty-six hours. Irrigation through the ear canal flowed out through the wound. Both the wound and the ear cavity looked well. There was no diplopia on looking up.

On October 28th there was a free purulent discharge from the right ear, but the pain had disappeared. The facial anesthesia had cleared up except that the right cornea was less sensitive than the left. The facial (seventh nerve) paralysis was still present but seemed to be improving. The patient was discharged from the hospital.

The postoperative course of this case was entirely uneventful with the exception of the involvment of the seventh, fifth, and a portion of the third cranial nerves. Healing eventually took place with a dry ear, but was considerably delayed by the undiscovered presence of a second piece of the defective rubber tissue which was used as a drain at the time of the operation. After the recovery of this fragment, the wound healed in less than a week.

The paresis of the seventh nerve, beginning seventy-two hours after the operation, and clearing up completely in the course of a few weeks, presents no point of especial interest.

The involvement of the fifth and that part of the third nerve supplying the superior rectus and the levator palpebrae superioris is a much rarer and more puzzling complication. A search of the literature for the past four years has disclosed only one case bearing any similarity to the one here reported: Auriti in the Atti d. Clin. Oto-Rhino-Laryngol., published by

the University of Rome in 1920, reports a case of acute right mastoiditis, with paralysis of the external oculomotor nerve (6th), neuralgia of the trigeminus, pupillary mydriasis. This case occurred in a man thirty-two years of age, following an acute catarrhal rhinopharyngitis and an acute right otitis media. Two weeks after the appearance of mastoiditis, diplopia on lateral glance developed, and neuralgia in the first and second branches of the right trigeminus. The case presented: 1, paresis of the abducens; 2, violent neuralgia of the first and second branches of the trigeminus; 3, persistent right-sided mydriasis with normal reaction to light and accommodation.

Dr. W. T. Coughlin, of St. Louis, in the Journal of the American Medical Association, December 15, 1923, calls attention to the fact that not infrequently, following section of the posterior root of the Gasserian ganglion, there is a paresis of the seventh nerve in those cases in which the motor root is severed, and that the face is drawn to the operated side in many of the cases in which the motor root is spared.

Various explanations are offered to account for the phenomenon above described:

First, that grand old refuge in cases not easily explainable, namely, hysteria. In the case here reported there was nothing in the family or personal history, or in the behavior of the patient, to justify any such conclusion.

Second, some disturbance taking place in the nuclei from which these nerves arise in the central nervous system. In view of the present state of our knowledge, or lack of it, in regard to the relationship existing between these nuclei, it does not seem to me that this theory offers a plausible explanation of the phenomenon under discussion.

Third, infection traveling along the connections between the two nerves, setting up a neuritis of these correlating branches.

Of the numerous connections between the fifth and seventh nerves, the structures in the carotid canal seem to offer the most probable pathway of infection in this case. Gray states that in this canal the internal carotid artery lies at first in

front of the cochlea and tympanus, and from the latter cavity it is separated by a thin, bony lamella which is cribriform in the young subject and is often absorbed in old age. Farther forward it is separated from the Gasserian ganglion by a thin plate of bone which forms the floor of the fossa for the ganglion. Frequently this bony plate is more or less deficient, and then the ganglion is separated from the artery only by a fibrous membrane.

Auriti, in his case, explains the symptoms as follows:

"The paralysis of the sixth nerve (which was not present in the case here reported) was the result of an infectious process, which progressed from the cavity of the tympanum through the pneumatic cells around the eustachian tube and the carotid canal to the apex of the pyramid, where it caused an osteitis or a pachymeningitis which involved the nerve at the point where it leaves the osteofibrous cavity, or in the canal itself. The neuralgia of the first and second branches of the trigeminus arose as follows: Infection starting from the middle ear reaches the carotid canal very easily through the caroticotympanic canal, which communicates between the canal itself and the antero-inferior portion of the tympanic cavity, finally reaching the middle part of Meckel's cavity, and from there the zone of the Gasserian ganglion that corresponds to it. In fact, the neuralgia strikes (1) the ophthalmic nerve, which has its origin in the internal part of the lower margin of the Gasserian ganglion; and, (2) the superior maxillary, situated externally to it. The irritation of the carotid plexus of the sympathetic clearly accounts for the pupillary mydriasis."

"We must, then," he concludes, "assume that the infectious process traveled along the carotid canal, where it caused irritation of the sympathetic, and reached Meckel's cavity through the inferior part of the canal, causing a focus of circumscribed pachymeningitis which was responsible for the neuralgia of the first and second branches of the trigeminus. It finally reached the apex of the petrous portion of the temporal bone, where it brought about paralysis of the abducens. Mastoidectomy resulted in complete cure of all symptoms."

A somewhat similar hypothesis would account for the symptoms in this case.

Because of the unusual amount of curetting which was necessary in the anterior portion of the tympanum, and the frequency of dehiscenses between that cavity and the carotid canal, it is quite conceivable that an infection traveled along this pathway, and with a possible deficiency in the canal wall forming the floor of the Gasserian ganglion, excited an inflammatory condition of the latter structure which manifested itself by tingling followed by anesthesia over the distribution of the fifth nerve.

The peculiar muscular reaction, namely, the deviation of the lower jaw to the left, and difficulty in swinging it to the right, would suggest that the motor root was not paralyzed but was irritated sufficiently to keep the muscles supplied by it in a state of contraction.

Whether the paralysis of the superior branch of the third nerve was affected through the sympathetic branch from the cavernous plexus or by its propinquity to the Gasserian ganglion, the writer ventures no opinion.

Another point of interest in this case is that it was a demonstration of the new radical mastoid operation presented by Dr. Barany at the meeting of the American Academy of Opthalmology and Oto-Laryngology, held at Minneapolis and St. Paul in September, 1922. The essential features of this operation is that the membranous canal is preserved intact and no plastic operation performed on this structure. The same care is used in chiseling away the bony canal wall as is used to remove bone which is adjacent to the dura. The treatment of the tympanic and epitympanic spaces varies with the pathologic conditions found. In some cases he recommends only the removal of the incus. In others, where the pathology is more extensive, the malleus is also removed, together with as much of the tympanic membrane as adheres to it. The removal of bone is identical with that of the usual radical operation, but the wound is closed and the antrum and mastoid cells allowed to fill up either with blood clot or granulation tissues, as in a simple mastoid. In the tympanum epidermatization proceeds and lines that cavity and the exposed attic, covering over the granulations which have filled the aditus ad antrum. One important advantage of this operative procedure is that a normal ear canal remains in which

there is no collection of epithelial debris, to be removed at stated intervals, as in the ordinary radical operation.

Since his demonstration, we have adopted this procedure in a number of cases, with most satisfactory results. Of these cases, I have no record of any in which the eustachian tube reopened. In other words, the ears have all been dry. The length of time it has taken our cases to heal has been longer than in the series of cases which he reported. Some of them have apparently been completely healed in two weeks, but others have required a much longer time.

As to the comparative effect on residual hearing, our cases do not offer a criterion, as none of them were able to hear the whispered voice more than six feet before operation.

XX.

THE UNSOLVED PROBLEMS OF OTO-LARYNGOLOGY.

By Harris P. Mosher, M. D.,

Boston.

The three preceding papers have given the accomplishments of otolaryngology to date. My subject is the unsolved problems. Fortunately, there are many of them, otherwise we should lapse into routine. We should degenerate into superficialists. To cover all the unsolved problems in a short paper like this one would make the paper hardly more than a catalogue of diseases. Therefore, I shall take only the larger problems.

As I see it, the two major unsolved problems in otology are chronic progressive deafness and meningitis. We can, of course, know more to advantage about brain abscess and intracranial pressure. Also we need to know the present day value of the Barany tests. As aurists, we are in a peculiar and embarrassing position. Patients come to us for the relief of deafness and steadily become more deaf under our care. Then they become the prey of the advertiser in and out of the fold, of the electrical and X-ray dabbler and of the osteopath. For some reason unknown to me they are seldom taken on by the Christian Scientist. We examine these patients closely for a possible focus of infection and in the majority of cases do not find it. In despair, we take out the tonsils and, as far as my experience goes, in most cases do not stop the progress of the infection; in fact, in a few cases the speed of the infection is apparently hastened. This has happened not only to some of my patients but to the patients of one of my confreres. We need sadly, therefore, the etiology and pathology of this pernicious low grade chronic progressive infection of the nasal, pharyngeal, tubal and middle ear mucous membrane. I have long been convinced that there are more mucous membrane changes than can be classified under the ordinary terms of hypertrophy and atrophy.

Speaking of infection, Emerson did us the great service of pointing out that in chronic progressive deafness, the toxin, whatever it is and whatever its source, in many cases affects the auditory nerve simultaneously with the involvement of the middle ear. This, to my mind, is a step in advance. Rosenau's theory of the selectivity of bacteria has never been judicially worked out by others. He has a few admirers and many who scoff at him. In this matter of deafness, however, we know too little, accomplish too little to sit on the scoffers' bench. Speaking of unsolved problems, here is a chance for some bright young man or for some older man who is still bright to do brilliant work.

This is the day of toxins. I have long felt that the accessory sinuses might be splendid places for the elaboration of toxins. Toxins can be produced without formation of pus. Serum can be highly toxic. Mucus can be toxic—that is, both sterile and toxic. I have long been suspicious of the white sterile masses of mucus which we wash from the antrum now and then. It occurs most often in vasomotor cases. I have had one unconfirmed observation made with this mucus. The patient had long suffered from a left trigeminal neuralgia. There was a history of a probable infection of the left antrum some time previously. I washed from this antrum about a dram of white sterile mucus. This injected into the peritoneal cavity of a guinea pig killed it within twenty-four hours. The abdomen and the rest of the body were negative at autopsy except for a slight congestion of the adrenals. In other words, this pig was overwhelmed by the toxin contained in the mucus. The usual control experiments were done, leaving the conclusion as given above. Is this anaphylaxis or toxemia? If this finding can be repeated in other cases, here is a new line of investigation.

Don't misunderstand me on this focal infection business. It is my stock in trade, the same as it is yours. But I cannot find the focus as often as some of you seem to do. My divining rod often fails me. I apply it to all parts of the body. I try never to forget that there are twenty-eight odd feet of intestine as a possible laboratory for toxins. As a routine, I give the patient the benefit of the fuller knowledge of the general practitioner. Nevertheless, chronic infection goes on in many

of my cases unabated, its source undetected. Sometimes I think that the otologist ought to change his name.

I expect help in this study of chronic progressive deafness from the new instruments of precision which enable us to record defects of hearing. We may be able to learn something about toxemia of the nerve of hearing through them. For instance, if a thousand painters were examined for the effect of lead, a thousand syphilitics, a thousand smokers, a thousand affected ears, and so on, we might have something more than our labor for our pains.

If only there was some quantitative way of measuring the hearing of animals. I almost said if only animals could talk. This, however, would be dreadful, because we should soon know what they thought of us, and the detective force of the world would be augmented by the whole animal kingdom.

THE ENDOCRINES OR THE DUCTLESS GLANDS.

Glandular therapy is on the crest of the wave at the moment. The newspapers are having fun with it, and tea table talk sidles up to it. The shotgun therapeutist keeps the slaughter house and the manufacturing chemist busy turning out polyglandular extracts. Swift succeeded by wasting nothing. He now wastes less. The monkey dies that we may live, or, rather, live again.

In spite of the tenor of the foregoing sentences, I am in great sympathy with this new form of therapy. It is simply an extension of new knowledge of biochemistry. Much of the future of medicine lies in the domain of this science. Lawrence of Boston, in a recent paper, opened my eyes to the possibilities of glandular therapy. He has greatly improved two cases of otosclerosis, one by correcting a deficiency of pituitary secretion and the other by ovarian extract. He has had some success also with cases of vasomotor rhinitis, cases, on the one hand, in which all the anaphylactic tests were negative, and cases on the other hand in which the patients seemed to react to everything.

The unsatisfactory thing about glandular therapy at the present time is the tendency to give polyglandular extracts without first determining what glands are at fault. Work in this line is still a sort of superscience, and but few men are doing it adequately. Rowe of the Evans Memorial, Boston,

has lately made an advance by perfecting a test by means of which he can pick out the gland at fault as between the thyroid, the pituitary or the sex glands.

Drury, also of Boston, is working under Rowe on the problem of otosclerosis. He has traced two cases of tinnitus to glandular insufficiency and stopped it by glandular therapy. Here, then, is a most promising field for research which deals with an unsolved problem.

In biochemistry the calcium content of the blood is looming large in its importance. The bone changes in otosclerosis have been compared to the bone changes of rickets. Rowe is working also on the calcium problem. In two years he hopes to have a method of computing the calcium content of the blood. The time he intends to give to the problem shows its complexity.

Now, how about meningitis? Are there any rifts in the clouds which surround this fatal disease? We know that some cases of meningitis get well, but how we do not know. We have learned that a cloudy spinal fluid does not necessarily mean bacteria in the fluid. Drainage and attempts at irrigation of the brain cavities and of the cavity of the skull are the modern methods of attack on this disease. Recently, in addition to blood therapy, methylene blue and mercurochrome have come in. Constant work is being done on sera. High hopes are held out in connection with a new serum for affections caused by the pneumococcus.

In operations we have two extremes—the subdural drainage of Crockett, and the occipital basilar drainage of Haynes. The first, in my opinion, has accomplished little and the second has failed. Day proved that the operation of Haynes would no more drain the pus in front of the brain stem than behind it. There are two places in meningitis where the pus accumulates, beneath the pia arachnoid quilted into the sulci of the brain and at the base of the brain, especially in front of the brain stem. In most cases it is the basal pus which kills. We cannot wash the sides and the base of the brain, and the arachnoid lakes of pus cannot be emptied by any single surgical procedure. You can wash the ventricular system of the brain very satisfactorily, washing from the lateral ventricle anteriorly to the fourth ventricle and the cistern posteriorly, and from the cistern to the lumbar region. It is very pretty to see this car-

ried out. Unfortunately, the ventricles are involved only secondarily and late in the disease. It does no good to wash only the inside of a dirty window.

There is a slogan, "Have faith in Massachusetts." There is also a slogan, "Too much Massachusetts." I will not speak of the latter. But in this matter of meningitis I would change the first slogan to "Have faith in Eagleton."

I have also faith in vaccines. In a recent case of mine, startling temporary benefit resulted from the intravenous and the intraspinal injection of a pneumococcus vaccine. I feel that serum treatment, combined with some sort of nasal drainage, offers our best hope of advance in the treatment of meningitis.

Now a word about the Barany tests. They should be evaluated anew. The red revolution which they promised in diseases of the labyrinth has not materialized. Eagleton has put the tests on the defensive by his showing the importance of increased intracranial pressure. Why are operative cases of suppurative labyrinthitis so uncommon of late? Have the Barany tests and the modern labyrinthine operation scared them off? Whatever the reason, in our hospital we have had very few labyrinthine cases for the past few years.

The manufacturers of the audiometer made the inspiring suggestion that with the type of deafness in any given case accurately charted by the new machine, it would be possible to write a prescription for a hearing instrument of the acousticon type which would correct the hearing defect. This promise has not been fulfilled as yet. The reinforcement of the residual hearing and the bridging of the islands of deafness might be accomplished in this manner—at least, it is theoretically possible. If we could write prescriptions for deafness the same as the ophthalmologist writes for glasses, we should all be sure of the monthly rent in advance, as he is. In fact, we could take another leaf from his book and turn his work over to a trained assistant, and give the time saved to other things—medical meetings, hospital work, teaching—in fact, to any form of medical luxury which appealed to us.

Operations for acute mastoid conditions are satisfactory in the great majority of cases. Here, for once, as aurists we shine. Heath, whose slogan was the self evident but forgotten fact that the ear was primarily an organ of hearing and that

early operation tended to preserve the hearing, was berated for some of his methods until the criticism stopped only from sheer exhaustion. In spite of his faults, he helped to put us back on the right track.

The radical mastoid operation also is satisfactory. Early resort to it cures a surprising number of case which have beginning labyrinthine symptoms. Sinus thrombosis is well under control in most cases, if the attending specialist is surgically inclined and not given to watchful waiting, and fond of making a diagnosis of typhoid. There is no such thing as typhoid in an ear hospital.

I have said nothing about the otologist as a brain diagnostician and surgeon. The young otologist should make himself a respectable neurologist. This done, his brain operating is limited by his opportunities and ambition.

The last two paragraphs tread a bit on the ground of the previous speaker, and show that I am coming to the end of my thoughts on the unsolved problems of otology. Therefore, I will stop here.

How goes it with laryngology? My first observation is that there are a number of disappearing diseases in the category of laryngology. Fibromata of the vault of the pharynx, at least in my part of the country, is getting more rare. I see fewer cases of papillomata of the larynx in children and in adults than I used to see. Tuberculosis of the larynx is also seen less often in our clinics. Whether this is true of tuberculosis centers or tuberculosis sanatoria I do not know. The ordinary throat clinic no longer considers itself the best specialist in such cases. The public may have found this out. Diphtheria is under control by antitoxin, and there is a campaign on foot to exterminate it through the aid of the Schick test. Should this come about, stenosis of the larynx would lessen in frequency.

We still have in nose and throat, as unsolved problems, the function of the tonsil, the cause of atrophic rhinitis, food deficiency, especially in children, as a cause of infection of the nasal mucous membrane, and suppuration in the accessory sinuses—in other words, the whole question of vitamines. The effect of vitamines on bone growth has not been worked out. This question is equally applicable to the ear.

The common cold remains an unsolved problem. The Surgeon General's office has taken up this subject as a peace time problem. All of us have received a questionnaire on this subject and few have filled them out.

The nose and throat are believed to be the portal of entry for many of the exanthemata. Some of the first signs of these diseases are seen in the mouth and throat. Why not begin the investigation of the causes of this diseases where they start —that is, in the mouth and throat?

Mullin has done good work on the lymphatic drainage of the accessory sinuses in animals. Good work also has been done on the lymphatics of the trachea. As the lymphatics are the chief highways of infection, these roads should be actively and continuously policed.

The actual value of diathermy in the removal of malignant disease, as well as the practical value of coagulation necrosis as a surgical advance guard, are two points upon which a judicial opinion is needed.

We have made considerable progress in the treatment of anaphylactic diseases, such as hay fever and asthma. Here again, however, the results are not uniform. Only a few cases comparatively are cured, and the question of bacterial toxins from the bacteria of the nose and throat is thrown back on us with renewed vigor.

Mudd, who started at Harvard and is now with the Rockefeller Foundation, is doing work on the nasal mucous membranes and bacteria which will bear watching. Sinus operations in laryngology are fairly satisfactory. We do pretty well with the antrum, the frontal sinus and the sphenoid, less well with the ethmoid. The overoperated nose, especially the overoperated ethmoid labyrinth, is worse, if possible, than the overoperated abdomen.

What form of tonsillectomy to advise in trained singers is still a question with me. The final effect on the fauces of the different methods of removing the tonsils has not been put on paper authoritatively. Is there such a thing as a dry throat after tonsillectomy and caused by it?

Infection of the internal jugular vein in the neck, or rather in the pharyngomaxillary fossa, after a subsiding tonsillar infection or in connection with a peritonsillar abscess or a retro-

pharyngeal abscess, is not clear to the mind of the general practitioner, to say nothing of its not being clear to the mind of the specialist.

Last of all, but most discouraging of all, is the subject of malignant disease of the nose and throat. Gratifying progress has been made in treating malignant disease of the accessory sinuses. We are now getting cures. Also, we are occasionally getting cures of cancer of the larynx. But what can we say of carcinoma of the esophagus? I have spent my whole medical life surrounded by this disease, almost pursued by it. I stand hopeless before it today, as I did when I first started practice.

In speaking of malignant disease of the accessory sinuses, allow me to reminisce just a bit. Resection of the superior maxilla was the popular operation when I was house officer. Gradually the nose and throat man with his head mirror and his special knowledge of the anatomy of the sinuses began to try these cases. Then radium came in, and operation on the sinuses was followed by the use of radium. The wound in the cheek was left open for radium treatment and in order to watch for recurrence. It was found that the cases which come to the throat department rarely had the floor of the antrum involved, so that it was unnecessary to connect the nose and the mouth. This in itself was a great step in advance. The results in these cases of malignant disease of the accessory sinuses are distinctly encouraging. For instance, in one series of nine cases I have had four cures, or rather four cases in which recurrence has not occurred, over a period of three years for one, three and a half, two, and four years for the fourth. It has been said that malignant disease of the accessory sinuses is less malignant than malignant disease in other parts of the body. This may be so, but the microscopic findings in these tumors do not always substantiate this statement. Less malignant or not, we are getting cures now, when formerly we did not. This question of the lesser malignancy of the accessory sinuses is one of the unsolved problems.

It goes without saying that the cause of cancer is still one of the most baffling of the unsolved problems of all surgery. The old question of the transmission of cancer by heredity has, to my mind, been proved, at least as far as mice are concerned. The findings on 20,000 mice, over a period of ten

years, have proved that cancer is transmitted to the offspring. The further suggestive observation was made that the inherited cancer always occurred in related organs.

In the treatment of cancer of the larynx, advance has also been made. The cases are increasing of cure of intrinsic cancer of the larynx by thyrotomy. More extensive cancer than is extrinsic cancer is being cured by total extirpation of the larynx. A few operators exclude the operation of thyrotomy entirely, relying in all cases upon extirpation of the larynx. How they draw the line in doubtful cases is still an unsolved problem—that is, at least to me. The after treatment of cases of extirpation of the larynx is being bettered. Still more work should be done along this line. At any rate, operators should acquaint themselves with the important work of Mackenty along this line before attempting the removal of the whole larynx.

Cancer of the esophagus remains the worst of the cancer problems, not only as far as laryngology is concerned, but as far as the whole body is concerned.

We have improved our methods of making the diagnosis of cancer of the esophagus. We can make these cases more comfortable. Radium, especially radium emanation in the form of seeds, has proved the most useful method of employing radium in these cases. I have yet to see a cure of a case of cancer of the esophagus. Dr. Green, who is connected with our cancer hospital, has had the same experience. Within the past two years we have had the use of the powerful deep X-ray. As yet no cures have resulted from this in our hands. In one case I have seen the histologic character of the growth change from malignant tissue to fibrous tissue. When the patient came for treatment, however, there was a question of an extension to the chest, and the patient finally died of this. We still have hopes of the therapeutic benefit of deep X-ray. What is the final value of this form or treatment is, therefore, one of our unsettled problems.

There have been at least two successful removals of carcinoma of the middle portion of the esophagus by the thoracic route in this country, one by the lateral chest route, and one—the case of Lilienthal—by the route through the back into the posterior mediastinum, and the carrying of a skin flap.

In the foreign literature something like ten or a dozen cures have been recorded in which the thoracic route was used. The upper end of the esophagus was brought out at the root of the neck and then carried down under the skin of the chest, later to be joined to the stomach by using a piece of small bowel to supply the lower half of the esophagus.

Here is an unsolved problem, if there ever was one in surgery. Here is a problem worthy of the mettle and the life work of any young man, surgically trained, who feels that the surgery of the nose and throat offers no problems comparable to the unsolved problems of general surgery. I, for one, should like to start over again with this problem.

To jump from the sublime to the odorous, let me say a word or two on atrophic rhinitis. We can mitigate this disease, but we do not know the etiology or the pathology. Two men with biblical names—Abel and Perez—have isolated organisms which for a time were thought to be the causative organism of the disease. Vaccines have been found to give only temporary relief. Narrowing the wide nasal chambers has seemed to mitigate the disease, but it is a makeshift procedure in comparison to finding the cause and setting about the cure in the logical fashion. Why the disease is self limited we do not know. The discovery of this point might help to put us on the right track. Here we sorely need some thorough, painstaking, scientific work. We need a relay of subsidized workers, because the cases are so unlovely that the workers must be skilled if the work is to be continued until something worth while is accomplished.

The bronchoscope has not fulfilled the promises of its youth as a means of enlarging our knowledge of thoracic diseases. The treatment of foreign bodies, lung suppuration, is as far as we go. Asthma does not yield readily as a routine to treatment by means of the bronchoscope. We occasionally make a diagnosis of malignant disease of the trachea or of the lungs by means of the bronchoscope. Except for one or two fortunate cases of this kind, we have gone no farther. We stand in about the same relation to malginant disease of the trachea and the lungs as we do to malignant disease of the esophagus. Coming back to diseases of the esophagus: The external two stage removal of esophageal pouches is a great advance in

safety. Cutting the common wall between the pouch and the esophagus through the esophagoscope has been proved efficient, but it has not yet been made safe. The treatment of single strictures is fairly satisfactory. The treatment of cardiospasm, due to stricture of the terminal portion of the esophagus, often yields brilliant results, as brilliant as any in surgery. It looks as if infection within the cone of the diaphragm, due to lower thoracic disease, will be found to be a cause of stricture of the terminal portion of the esophagus in a certain number of cases. Basal tuberculosis alone, associated with tuberculosis of other parts of the lungs, has lately become another possibility as a cause of stricture of the lower end of the esophagus. The cause of stricture of the lower end of the esophagus, therefore, is another of the unsolved problems waiting for the investigator.

The best method of treating nuts in the trachea or bronchi of young children may be a solved problem to most of you. It is not to me. Unless the foreign body is just below the vocal cords and is readily extracted, I feel that tracheotomy is the best procedure.

The value of tracheotomy as a means of draining the lungs —especially if a punch tracheotomy is used and no tube is inserted—is not yet realized.

If the laryngologist does thyroid surgery, he must be familiar with the problems of metabolism. The major problems of metabolism are as yet unsolved. Speaking of thyroid surgery reminds me of the procedure of Frazier—that is, the suturing of the recurrent laryngeal nerve to the descending branch of the hypoglossal nerve. Here is another unsolved problem which seems on the way to solution. Any laryngologist with adequate surgical training should be able to contribute to the solution of this problem.

Status lymphaticus is still an unsolved problem. We are attacking this problem through the help of the X-ray. A thousand cases of tonsils and adenoids have already been X-rayed before operations. Six per cent of suspicious thymus glands have been found.

The value of X-ray therapy in the treatment of hypertrophy of lymphatic tissue is still under investigation. The effect of X-ray treatment on lateral pharyngitis and blocking of the

eustachian tube is in process of determination. No one has as yet succeeded in locating a brain abscess by means of the X-ray. Up to now the X-ray has indicated pathology only by the presence of shadows where shadows should not be normally. In the future, the X-ray should show bone changes like decalcification destruction. It is conceivable that it will get beyond shadow pathology in the interpretation of diseases of the mucous membrane.

In order to deal successfully with the unsolved problems, we need pathologists, men up in biochemistry, in immunity, and in serology. We need men to bring the advances in these subjects over into the specialty for use there. We need men trained along these lines, because they think large. The petty thinker seldom thinks beyond his specialty—I almost said, beyond his nose.

UNFINISHED BUSINESS.

Under the heading of unfinished business, the following subjects might be mentioned:

The problem of retrobulbar neuritis is a common problem, shared by the ophthalmologist and the rhinologist. The ophthalmologist should find a way to tell us which cases are self limited and get well of themselves and the cases which threaten the sight. White deserves great credit for sticking to this problem. His anatomic observations give something to pin to which we have not had before. Long may he live, prosper and work.

Meckel's ganglion complex has been long enough before the medical profession for somebody to make something of it besides Sluder. We need a neurologic follow-up on these cases.

Fetterolf should finish up his work on the etiology of lung abscess. I suppose that it is expecting too much of human nature to expect any committee appointed to investigate the tonsil problem ever to finish its work and to report.

State lye legislation, the credit for which is due to Jackson, should be pushed in all States. The problem of the standardization of the training of the specialist and a national examining board in otolaryngology is being pushed forward by Shambaugh. He divides his time between this and the tectorial membrane. The work on both, when completed, will stand as a monument to his sagacity, energy and diplomacy.

I have said nothing about psychoanalysis in otolaryngology. It must be apparent by this time that there are enough unsolved problems in otolaryngology to satisfy the ambition of anyone. Allow me, in closing, to be a bit sentimental and to say a word about the joy of work. The joy of conquest needs no comment. Some of you have known it. Most of us, however, have to be content with the joy of work. In my opinion, there is no pleasure so great as having your little try at the solution of some medical problem. Most men, to be sure, accomplish only millimeters of progress. The genius is the meter man. Remember, however, that a millimeter may be a day's journey for a microbe.

How can research work best be done; how can these unsolved problems which we have been discussing best be approached? It must be admitted that the business of getting a living is a nuisance. The genius gets on somehow almost without a living. This is part of being a genius. For the rest of us, research work can be done only after we have acquired a fortune early, like Dean, or hired ourselves out to some hospital or university, as I have done. It has been said, over and over again until the phrase is threadbare, that research laboratories should be endowed, otherwise research workers must have independent means. It is the same old question of money. We are called commercial if we speak of money. Money, however, may be made the most useful thing in the world. Even the poet must have it. When he pretends to scorn it, you will find that he lives, as Whittier did, for the most part on his relatives.

One almost becomes a socialist when he thinks much about this question of the adequate support of research work. Support or no support, research work has been done for years, on the side, as it were, and probably always to a certain extent will be done in this way. You and I have so done it. Better men than we are will do the same. The urge to do it has come down the ages. Here we are back again to the subject of the joy of striving. The worker is not to be pitied. Rather he is to be envied, because, given a "companioning ideal" and a correlating mind, the world of medicine becomes for him a world of romance which, translated into the vernacular, means get busy on the solution of some of these problems.

XXI.

HISTORY OF THE SINUSES.

By Ross Hall Skillern, M. D.,

Philadelphia.

When one is delegated to write a history of anything, in order that it should be considered reliable he must observe two principles. First, accuracy of statement, and second, credit given to the proper individuals. How this can be done in this instance without boring the audience to tears is a problem which I will not attempt to solve.

Accuracy of statement in the history of medical affairs is usually so commonplace that to designate it as "drab" would, to my mind, fall little short of actual flattery. How much more interesting it is to believe, for example, that standing bareheaded in the shadow of the tomb of Lafayette, Pershing repeated those historic words, "Lafayette nous sommes ici," when as a matter of fact he did nothing of the sort, but remained silent while one of his aids whispered these words with all the inflection and pronunciation of a born Parisian. Such, however, is history. To be of absorbing interest, romance must hold sway, and where one can find anything pertaining to the romantic in the accessory cavities of the nose is beyond my comprehension.

With this in mind, we shall now settle ourselves to our task. It would appear that the first mention of the sinuses was made in a rather abstruse manner by Hippocrates, when he said, "In a person having a painful spot in the head, with intense headache, pus or fluid running from the nose removes the disease." This, I take it, must have been a purulent sinusitis or else some disease which was known only to the ancients and has become as extinct as the proverbial unicorn.

Coming down through the ages, it was probably an Italian anatomist named Berengar who taught surgery at Bologna from 1502-1527 that was the initial one to definitely indicate their existence,[2] although Galen is said to have referred to

the porosity of the bones of the head. Vesalius[3] first described the individual sinuses, and his work was further augmented successively by Fallopius,[4] Veslingius, Jessen and others who also theorized on the functions of the sinuses. Some of these conjectures were quite curious, as for example that they contained a humor with which the eyes were moistened and lubricated. Another, that they are for the retention of odor before it is carried to the brain. Veslingius,[5] in 1637, said, "There is much doubt as to their use." I wonder if these eight words are not as up to date today, in 1924, as they were when uttered some three centuries ago.

Much might be said of those who, little by little, added to our knowledge of the sinuses, but time is pressing, so let us now come into the era which saw the birth of modern sinuology. Zuckerkandl of Vienna, a professor of anatomy, facetiously known in England as Professor Sugar Candy, by his researches on the nose and sinuses[6] gave us our first comprehensive and intimate knowledge of these structures, and it was on this solid foundation that the specialty was founded. Although preceded by a number of isolated observers, Grünwald[7] of Munich was the first one to write a treatise on suppurative sinus disease, which, though well conceived, was badly compiled, so that one was obliged to read much in order to gain little. Hajek of Vienna, two years later, brought out his first edition of the most famous book on the sinuses that has ever been written.[8] Indeed, it is my opinion that this book was in a considerable measure responsible for the great repute of Vienna as a nose and throat center, and this, coupled with Politzer, on the ear, made that city the Mecca for American specialists during the halcyon days of the nineties and early years of the twentieth century. About this time a Badenese laryngologist, who resided in the Black Forest, named Killian, began to attract the attention of the laryngologic world by his researches, so that he soon became internationally famous, and what Hajek was to the Austrians, so Killian was to the Germans. This soon resulted in a personal rivalry, which ended in a polemic (which is only a polite synonym for a war), and as the Germans and Austrians both took their respective sides, the lines became sharply drawn, and neither would Hajek attend laryngologic conventions in Germany nor

would Killian come to Vienna. This impasse was, I believe, broken when Killian attended the International Laryngological Congress in Vienna in 1908. In France and England, men were coming on who were destined to become masters, such as Luc of Paris, Logan Turner of Edinburgh and Tilly of London.

But enough of this, as we must turn to our own country and this society, for such, after all, was the purpose of this dissertation. Although the subject was considered before this, the first reference to the sinuses was made by Beverly Robinson of New York, in 1886, his subject being "Inflammation of the Antrum."[9] He believed all infections of this cavity were due to diseased teeth and advocated the extraction of the teeth and opening the antrum through the socket as the one method of treatment. The next important treatise on this subject was Bosworth's essay on "Various Forms of Disease of the Ethmoid Cells," appearing in 1891, which opened up an absolutely new field, being in the form of a sensational revelation.[10] This observer attempted to classify the pathologic conditions of the ethmoid, and while he considered certain conditions, as, for example, extracellular and intracellular polyposis, as separate diseases instead of an advanced stage of the same, nevertheless, he was really the first one to recognize a difference between suppurative and hyperplastic ethmoiditis. Shortly after this, Woakes of London wrote his famous paper on a condition described by him as necrosing ethmoiditis.[11] His British colleagues did not take kindly to his nomenclature and denied that such a condition existed. The issues of the British Medical Journal of December, '92, and January, '93, contained many letters and statements disputing his findings. Zuckerkandl,[12] in particular, was most outspoken when he said, "The statement of Woakes, which, on account of the operative consequences that might ensue, cannot be too sharply combated, and every rhinologist should be warned that the socalled condition of necrosing ethmoiditis came more from the depths of his imagination than from original scientific research."

The following year a Dr. Rankin of Allegheny, Pa., presented a paper on Diseases of the Frontal Sinus,[13] and called attention to its frequency following attacks of la grippe. In 1895, sinusitis was quite generally recognized, and the pro-

gram of the American Laryngological Association for that year contained papers by Bosworth, Bryan and Knight. In 1897, Bryan of Washington reported probably the first case performed in America of the external operation on the frontal sinus with drainage through the nose.[14] The method used was a combination of Ogston's and Luc's. The latter method appeared the year previous. In the same year Holmes of Cincinnati brought out the relation of the sphenoid sinus to orbital conditions, a paper which made a profound impression by its evident importance. Myles, in this society, also made an important contribution to the study of sinus disease,[15] and the following year Holbrook Curtis, also in this society, reported on the negative value of transillumination in antrum disease, a paper, however, that apparently has been sadly overlooked.[16] The first attempt at a symposium was held by the A. L. A. in 1899, and was on "Diseases of the Ethmoid Region and Asthma." The presentations were made by Swain, Rice, Ingals and Bosworth, while the discussion was carried on by the elder Shurly, J. N. McKenzie, Hudson Makuen, Casselberry and Hubbard. Most of these men have gone to the great beyond, and their names are now familiar only through the literature and traditions of our specialty. Several other papers on the frontal, maxillary and sphenoid were read, and the first report in America of results following the Caldwell-Luc on the antrum by one in our calling was read by De Roaldes of New Orleans. A new phase of sinus disease was brought out by Emil Mayer in 1901 on Maxillary Sinus,[17] and Hinkel, the following year, with his treatise on the sphenoid,[18] was the first American laryngologist whose work was recognized in German literature.[19] In 1903, Luc of Paris visited this country, being the guest of honor of the A. L. A., and gave a dissertation on the radical treatment of sinus suppurations.[20] That the importance of the sinuses had now taken hold and was becoming generally recognized was further evinced by beginning original investigations in both the pathologic laboratory and deadhouse. It was at this time (1904) that Mosher first came into prominence with his thorough and painstaking researches into the anatomy of the frontal sinus.[21] This work at once become the standard guide for the intranasal operator and has remained so ever since. In the early summer, Logan

Turner of Edinburgh, being a guest of the A. M. A. at Atlantic City, read an exhaustive paper on the different external operations on the frontal sinus.[22] I recall this vividly, as this meeting was the first one that I ever attended, and I can well remember the feeling of awe and deference which possessed me while listening to the essayist treat with such familiarity the intricacies and mysteries of this to me unexplored and unknown region.

At the meeting of the Triological Society, Coffin[23] reported the results of operations on children for sinusitis, and his name has been indelibly associated with sinusitis in children ever since. The following year (1905) seems to have been a banner year for this comparatively new study, for in addition to the first real symposium on the sinuses held by the A. L. A., two candidates' theses were upon this subject, Coffin of New York, who spoke of the operative phase of frontal sinusitis,[24] while Richards of Fall River went into some detail as to the clinical aspect.[25] A few weeks later, at the meeting of the A. M. A., at Portland, Richards presented in detail the intranasal method of operating beneath the inferior turbinate for maxillary sinusitis.[26] Fletcher Ingals of Chicago also brought out his intranasal operation on the frontal sinus, accomplished by means of an engine driven flexible burr.[27] I think no one else had the temerity to attempt this operation, owing to the double danger, the proximity of the anterior portion of the cribriform plate to the rapidly revolving burr and the flexible armature. One thing, however, must be said, and that is that Ingals preceded Halls of Berlin by something less than two years, and the latter's method, I understand, is still in use in his clinic. A symposium was also held by this society, consisting of eight papers, which, however, emphasized more particularly the operative side.[28] Two years later (1907) Loeb of St. Louis presented the results of his anatomic studies on the sinuses.[29] This proved to be but the first of a series of these studies which made this investigator the Onodi of America. Onodi and Loeb —two names which must be always connected with the minute anatomy of these cavities.

The year 1908 seems also to have given the sinuses sway, as the A. L. A. held another symposium, where Gordon Wilson of Chicago presented his thesis on the importance of the

ostium frontale in its relation to intranasal procedures on the frontal sinus,[30] which has since become a standard for the sinuologist in operating in this region. At the Chicago meeting of the A. M. A., the same year, much time was devoted to the sinuses. Canfield brought out his operation on the maxillary sinus, which, in the discussion, met with considerable opposition, but it afterwards developed that nobody had quite understood the technic. Beck proposed a new method on the frontal, and the sphenoid was treated both from the anatomic and surgical aspect. (Jansen of Berlin was the guest at this meeting, and he certainly left an impression, but it was a bad one.)

The year 1909 saw the light of the intranasal rasp operation of Worthington on the frontal sinus,[31] although Good[32] had advocated this method the year previous. Thompson,[33] however, a year later brought the method to its present degree of applicability. Vaccine treatment for sinusitis was advocated in 1910 by Birkett[34] and Coakley.[35] Many of us here present can recall how this was to be the long looked for panacea, but, like the X-ray as an infallible diagnosis, so was the vaccine to prove most disappointing, until now its use is limited to certain selected cases.

More attention was being paid to the sinuses from the standpoint of the ophthalmologist, and many papers and cases were presented by our brethren in that specialty. Most of them recognized and advocated early operative interference (on the posterior sinuses), but some unfortunately actively combated this view as being ultraradical and unnecessary. We all know now which group had the proper foresight. Deaths following intranasal sinus operations were now beginning to be reported.[36] A circumstance which caused many more inquiries to be made and much more anatomy studied than was formerly the case. The market on dried specimens of the nose and sinuses became suddenly cornered. The pathology of ethmoiditis following Uffenorde's work[10] was being investigated and discussed, and I recall it was in this very city, after the essayist had endeavored to illustrate the microscopic differences between hyperplastic and suppurative ethmoiditis with lantern demonstration, the lantern persistently refused to function and

finally fell with a terrific crash to the floor, Ballenger exclaimed, "That's where it belongs; let it lie."

The complications of sinusitis, particularly those referring to the eye, now began to attract the special attention of the sinuologist (1911). In the section of the A. M. A., Sauer, Andrews and Bryan held almost a little symposium on the subject, while in this society Harmon Smith spoke of cases of optic neuritis benefited by operation upon the posterior sinuses.[37] By this time (1913) the sinuses were being recognized by the up to date general practitioner, and one no longer spoke in the abstruse manner when consulting with one of these and suggesting that the sinuses might be the seat of the trouble. Works devoted entirely to the subject were appearing in the English language, and in the various medical colleges the fourth year medical students were discoursing quite learnedly of the sphenoethmoid recess. In the dispensary patients, one always saw a potential sinusitis, and lucky indeed was the individual who got away without having at least a needle stuck into his antrum. This was also perhaps the heyday of radical operations on the sinuses, for the pendulum had swung far out and had not yet begun to return. New phases incident to the sinuses were coming into being, such as Sluder's sphenopalatine syndrome (1914). Mosher[38] brought out his operation on the ethmoid based on his anatomic studies. This operation has been either much maligned or blindly followed, few apparently taking the middle of the road. Both factions, I think, are wrong. The operation has its distinct place and has become a standard one in certain types of cases. Sinusitis in children was now attracting much attention, and indeed was considered of sufficient importance for the A. L. A. symposium, which was conducted by Coffin, Wood and Coakley.[39] The Killian operation on the frontal sinus did not appear to be giving general satisfaction, and those using it had gradually become convinced that such an extensive procedure was not always necessary, and the results ofttimes were by no means all that had been hoped for. Other methods had been devised but none met with universal approval. Lathrop (1915), a general surgeon, proposed a special procedure which was quite different from anything before attempted, being in the nature of a small external opening with large internal drainage.[40]

The operation met with instant approval until it was attempted, then the difficulties at once became apparent. I do not believe many of our brother specialists are now performing the method as originally advocated by Lathrop. This brings us up to 1916. Extensive intranasal operating upon the ethmoid was not generally practiced, but was really confined to the hands of comparatively few men. The reason for this was the difficulties and dangers, for, as Mosher once said, "Ethmoidal operating, which deserves the name, always has been dangerous and always will be." No truer words were ever spoken, and I can add that the danger is tripled if done under general anesthesia. Sluder evidently had this in mind when he proposed an intranasal procedure in which he stated the cuts were always downward and forward.[41] While this method did not revolutionize ethmoid surgery, nevertheless, being based on sound anatomic principles, its influence was felt in the diminution of haphazard surgery in this locality. The war has now brought us into the maelstrom, and most of the profession were engaged in preparing either themselves or others for combat service, so scientific research was for the time being relegated to the background, and the medical profession as a whole devoted itself to the successful termination of the conflict. Dean, however, had been devoting much attention to sinusitis in children, and in the early summer of 1918 presented the results of his observations.[42] He showed that diseased tonsils and adenoids were frequent etiologic factors and their removal often brought about a cure of the sinusitis. While this does not obtain in every case, nevertheless whenever sinusitis is present in a child, a tonsillectomy and adenectomy should be performed as a preliminary procedure.

Orbital complications from the sinuses, particularly the posterior ethmoid and sphenoid, were now recognized by everyone, but making a special study of retrobulbar neuritis, White of Boston brought it into special prominence by giving us concrete data and making definite statements as to the advisability of immediate opening of these sinuses, whether or not symptoms pointed to them.[43] This dictum, if followed out, will be the means of saving unnumbered eyes in the future. A fine piece of original investigation on the lymph drainage

of the sinuses was carried out by Mullen.[44] The importance of this work will grow as time elapses, as already certain internists have made it a basis for their investigation. We are now approaching 1921. The etiology, pathology and treatment of sinus disease had long since been placed upon a firm footing and had become practically standardized. It is true certain modifications in operative procedures had been suggested, curious cases reported, some quite sensational ones, in which vision had been restored in the totally blind by opening the sphenoid, but on the whole little of absorbing interest had occurred in the last two years. And so it remains up to the present. The growth of sinuology from the tooth infected antrum, to be recognized as the causative factor of disturbed conditions in remote portions of the body, has been a comparatively rapid process in which American medicine has played no small rôle.

Such, gentlemen, is an exceedingly superficial review of our work on the sinuses.

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XXII.

CERTAIN NEUROOTOLOGIC PROBLEMS.

By Samuel D. Ingham, M. D., and Isaac H. Jones, M. D.,
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The purpose of this paper is to give a brief discussion of each of the main subjects included under neurootology. Its point of view is mainly clinical. The writers have made certain experiments in physics and with animals; but these are mentioned only to the extent that they throw light upon clinical problems.

The subjects to be considered include experimental research; clinico-pathologic research; auditory problems; endolymph movement; mechanism of ocular movement; vestibulo-ocular mechanism; quick component of nystagmus; spontaneous vertical nystagmus; perverted nystagmus; vestibulo-cerebello-cerebral tract; cerebello-pontile angle tumor phenomenon complex, vestibular and auditory; and neuraxial differentiation of vestibular pathways.

In summary, an attempt is made to answer the following specifications bearing upon the subject matter presented in this paper:

(a) What we are to regard as purely theoretical;

(b) What we are to regard as highly probable;

(c) What we may regard as reasonably proven, including the phenomena on which we can rely in clinical diagnostic work.

EXPERIMENTAL RESEARCH.

Experimental research has certain advantages over the clinico-pathologic, presenting as it does the opportunity either to stimulate or to destroy certain tissues, and then to study the results. These results can be studied by noting changes or loss of function, and also alteration or impairment of reactions to stimuli. In addition, it is then possible, by postmortem examination, to make gross and histologic study of the structures affected. The further advantage of such experimental research is the opportunity afforded to establish a series of ob-

servations and to obtain a general average of results from many similar experiments.

Much important knowledge of the vestibular apparatus has already been attained by the experimental method. This includes observation and classification of the responses which can be obtained from a direct stimulation or destruction of separate structures within the internal ear. S. S. Maxwell has made recent valuable contributions to this phase of the subject, by experimental work on fishes. Another result of experiment work has been the tracing of degenerated nerve fibers from the eighth nerve and its connections, in various directions within the brain stem and the cerebellum. The most recent and valuable work on this subject is that of Sachs and Alvis.

There are many difficulties peculiar to vestibular study, that are met by one undertaking experiments upon animals. There is the difficulty of applying the principles of comparative anatomy and physiology, due to the variations of structure and function in the different species. For example, the range of complexity of ocular movements varies greatly in different animals; vestibular stimulation which causes rotation of the human eyes in the frontal plane, produces in fishes an upward deviation of one eye and a downward deviation of the other. Caloric stimulation of cats fails to produce some of the responses obtained in the human being.

One of the most reliable methods of tracing the course of nerve fibers is by means of the degeneration which follows their injury. Such degeneration can be traced only in the distribution of the particular fibers injured. Although, physiologically, nervous impulses pass from the terminals of one nerve fiber to another neuron, and so on to their ultimate destination, pathologically considered, each synaps limits further degeneration. To repeat, degenerations can only be traced within the limitations of a single neuron—always being limited by the synaps. Many different pathways, such as that of common sensation from the skin, are known to include three or more successive neurons, including as many synapses through which the impulse must pass. To follow this pathway by the process of tracing degenerations, it is necessary to produce a lesion in each successive neuron. For example, the sensory pathway for

pain extends from the periphery to the posterior root ganglion; other fibers pass from there and enter the spinal cord; a short distance above the point of entry, connection is made with another neuron, the axis cylinder of which proceeds up the cord and through the brain stem to the optic thalamus. Here, in turn, new connections are made; the ultimate anatomic connections are as yet undetermined. The different forms of sensation, such as heat, cold, tactile, pressure and muscle sense, are each represented by a special neural pathway which can be traced over successive neurons along its course. Some proceed up the spinal cord on the same side that they enter, some on the opposite side; some are interrupted within the cord shortly after their entry, whereas others proceed without interruption to nuclei in the medulla oblongata.

Analogous arrangements may be noted in the nervous mechanism of the special senses. The retina, for example, contains within its layers not only the end organs, represented by the rods and cones, but neurons interposed between these end organs and the neurons which proceed in the optic nerve. Furthermore, the primary centers of vision form another interruption in the course of the visual pathways before they reach the cerebral cortex.

There are only a few demonstrated synapses along the vestibular pathways, such as Scarpa's ganglion and Deiters' nucleus; however, it is fair to assume, from the analogies just given, that there are numerous other breaks in the continuity of the neural vestibular pathways.

From the well established principles of secondary degenerations, it is readily seen that any part of any pathway can be traced only within the segment which has been injured, and that to trace any particular pathway throughout its various segments it would be necessary to produce lesions such as to involve each segment in turn.

In applying this method to the present problem, there arises the practical difficulty of making discrete lesions. To be of value, experimental lesions must be limited so as to destroy a small amount of tissue, and must be so located as to produce the desired destruction without disturbing adjacent tissues. In vestibular studies, it would be highly desirable to produce such minute lesions within Deiters', von Bechterew's and the

triangular nuclei; the cerebellar nuclei, globossus, emboliformis and fastigii; the corpora quadrigemina and many other structures. The difficulties attending such attempts are obvious.

To give concrete applications of the above general principles, we might consider the possible results to be obtained by destruction of successive segments in the vestibular pathways—(1) End organ, (2) eighth nerve, and (3) central nuclei.

1. End Organ.—The exact histologic course of fibers from each semicircular canal could be demonstrated if one canal could be destroyed and its pathways traced. (The same could be applied to the utricle and saccule.)

It is possible to destroy one horizontal canal and its ampulla, in an animal previously proven to be normal by caloric stimulation. To prove that only the horizontal canal has been destroyed, it would be necessary subsequently to demonstrate normal function in the vertical canals. This proof is probably impossible because of the serious disturbance of the entire mechanism of the end organ incident to the destruction of one canal. This would seem to be corroborated by the experiments of Sachs and Alvis, in the lack of uniformity of the spontaneous nystagmus which they observed, in the different animals in which only one canal had been destroyed.

Even if one canal could be destroyed and the fact of this isolated destruction proved by the caloric test, the secondary nerve degeneration does not extend throughout the physiologic distribution but stops at Scarpa's ganglion. Sachs and Alvis have demonstrated that destruction of all the canals is not followed by degeneration of the eighth nerve.

2. The eighth nerve can be destroyed and secondary degeneration traced to different portions of the brain stem and cerebellum. However, it has not yet been possible to determine any relationship between any portion of these fibers and the individual semicircular canals, or the utricle or the saccule.

3. Central Nuclei.—Isolated destruction of the central nuclei is much more difficult; and yet, to correlate the different nuclei with the different portions of the end organ, such a method seems to offer the only possibility for future study. In the work already done, in making discrete lesions in the nuclei of the brain stem, so far as we know only the spontaneous phenomena have been observed. Additional information might

be gained not merely by observing the spontaneous phenomena but by studying the responses to the caloric test.

If, for example, Deiters' nucleus were destroyed and subsequently the horizontal canal failed to give normal responses, whereas the vertical canals did produce normal responses, we could reasonably conclude that the fibers from the horizontal canal go to Deiters' nucleus, and that the vertical canals fibers do not go to Deiters' nucleus. So with each of the other vestibular nuclei—the von Bechterew's and triangularis—if it ever becomes possible to make discrete lesions of these structures.

Summary.—To find out the exact course of the fibers from the different semicircular canals appears to be impossible from experimental work on the end organ or eighth nerve. If ever this is to be attained, it seems that the only possibility consists in making discrete lesions in the brain stem and then testing the different semicircular canals by the caloric test.

CLINICO-PATHOLOGIC RESEARCH.

Clinico-pathologic study has its own peculiar advantages, among them:

1. The human being is the object of study. This eliminates certain fallacies always present in animal study. Studies of the human being are furthermore more complete, in that it is possible to secure the cooperation of the patient in making more elaborate tests than are possible in animals. There is another important advantage in that the human being can supply intelligent evidence of his subjective sensations, both spontaneous and after experimental tests.

2. Certain pathologic processes result in small discrete lesions, in locations within the central nervous system, which cannot be duplicated by operative work on animals. This includes vascular lesions, such as thrombosis and small foci of encephalitis and multiple sclerosis. Incidentally, lesions of this type give more exact information than tumors or abscesses, in which pressure phenomena may obscure the picture.

3. Clinico-pathologic study has the advantage of having its workers constantly pressed by the need of accuracy in diagnosis. Every clinician feels the necessity of accurate interpretation of phenomena which he observes in everyday practice. There may be, in the world, a dozen or a score of experi-

mental investigators interested in neuro-otology. In contrast, every otologist and every neurologist is a potential investigator in clinico-pathologic research. Furthermore, problems have been raised by clinical studies which have stimulated experimental anatomic studies. The ideal, of course, is to obtain exact histologic knowledge of each minute detail. In the meantime, the clinician can serve two purposes: first, to learn what he can from clinical cases, or operation or necropsy; and second, by his daily clinical observations to raise questions and present problems for the anatomist to try to solve.

In discussing experimental work on animals, it has already been mentioned that normal responses to ear stimulation should be made a basis of observations. It may be said that clinical experience with human subjects has already fairly established the norm. We know, with hardly any reservation, that when any normal individual is turned or his ears douched, there is produced a definite eye movement of a certain duration in a definite plane; a definite vertigo of a certain duration; and a falling in a definite direction. If he be turned or douched to an extreme degree, he also shows pallor, sweat and nausea.

With the above phenomena we become familiar, and all who undertake such tests become impressed with the uniformity of responses. Each variation from the norm must receive due consideration. To be sure, a certain latitude must be given to individual variations within the norm; when, however, gross deviations from the norm present themselves, it may be concluded that some portion of the complicated mechanism has been disturbed. It might be well to cite briefly certain examples of clinico-pathologic experience in patients that proved to have intracranial lesions.

1. In one patient ear stimulation produced normal responses from the horizontal canal and no responses from the vertical canals. Another patient showed the reverse—normal responses from the vertical canals and absent responses from the horizontal. Other cases, such as angle tumors, have shown no response from any canals of either ear, except one horizontal canal, and this showed entirely normal function. Two cases recently seen (Exhibit A and Exhibit B), not as yet pathologically verified, showed normal responses from all semicircular canals, except one horizontal—the exact opposite of the phe-

nomenon complex of the angle tumor. We conclude that there is a neuraxial differentiation—that the fibers from the horizontal canal are sufficiently separated from those of the vertical canals to permit either to be involved without any involvement of the other. This will be taken up in detail under the separate "Problems."

2. Comes a patient who shows normal nystagmus, but impaired vertigo on stimulation of one horizontal canal. Another shows normal nystagmus and impaired vertigo from both horizontal canals. Another shows normal vertigo but impaired nystagmus. We are led to consider that from the horizontal canal there is a further division of the pathways, on the one hand producing the nystagmus and on the other hand producing the vertigo.

Clinico-pathologic observations of lesions in different intracranial locations have shown such different phenomena that to explain them merely on the basis of general pressure is untenable.

In spite of the limitations in experimental and in clinico-pathologic research, each method has its own peculiar advantages and in many ways supplements the other. Further progress will be determined largely by the measure of cooperation of the investigators using the different methods of approach.

PROBLEMS.

To the clinician the problem is to evaluate each phenomenon observed in the study of patients. We will take up separately a number of debated subjects and will present, in skeleton form, certain evidence "against" and "for" previous conceptions of their significance.

It will be noted that the subject of forced movements of the skeletal musculature is not included in these problems. Magnus and de Kleijn and others have demonstrated physiologically that there is a lower circuit from the internal ear to the skeletal muscles—presumably via the vestibulo-spinal tracts. It would seem that these vestibular impulses are eventually distributed by spinal coordinating mechanisms in control of the musculature affected. As to past pointing, as tested by the Barany method, although it is essentially an adjustment to the conscious sensing of movement, yet it is probable that tonetic

influences through the lower pathways reinforce this adjustment. The falling response to ear stimulation appears to be essentially a forced movement, although there is a definite cerebral element. There is, however, so much uncertainty as to the clinical interpretation of forced movements that we have not attempted to present this subject as a problem in this purely clinical paper.

AUDITORY.

In the past fifty years very little has been accomplished in auditory studies. Recently, however, there has been a renewed interest and activity. Instruments of precision have been devised, the most promising of which utilize the audion bulb or vacuum tube. At the present time one can only speculate as to the value of these recent studies. However, it seems probable that new information will become available concerning the physics and physiology of the cochlea; lesions of the cochlea and eighth nerve; the significance of paracusis and diplacusis; and the phenomena of tinnitus.

As to the course of the pathways between the entrance of the eighth nerve into the brain stem and the cortical centers for audition, clinico-pathologic evidence is singularly meager. A priori, it might be expected that various brain stem lesions would exhibit an impairment of hearing; in clinical practice, however, hearing impairment from brain stem lesions is rarely observed. The explanation advanced for unimpaired hearing in cases of brain stem lesion is the neuraxial differentiation of the auditory fibers, by means of which each eighth nerve is in relation with fibers on both sides of the brain stem. Some impairment of auditory function might well be expected in brain stem lesions; in the present status of our knowledge, however, auditory tests are of practically no value in giving evidence of lesions within the brain stem.

It is possible that the newer instruments of precision may lead to the detection of central lesions. Two cases now being studied by Prof. Vern O. Knudsen and the writers, suggest such a possibility. Each case exhibits unilateral impairment of hearing. With noise apparatus in opposite ear, loud shouting cannot be understood. Yet on testing the complete tonal range, each case shows marked impairment for all lower tones up to a certain pitch, beyond which all tones of the upper scale

are heard with almost full normal acuity. At the present time the diagnosis is undetermined in either case. Such a phenomenon is unlike the usual case of perceptive deafness. It is possible that the outcome in these two cases may reveal whether such a phenomenon is caused by a lesion within the cochlea or a tumor of the eighth nerve.

Attention might be called to a distinct neuromechanism of the peripheral organ of hearing—the mechanism controlling the tensor tympani and stapedius muscles. These two muscles are so arranged that they appear to be concerned with the adjustment of various degrees of tension of the drum membrane and the ossicles. These adjustments are made to accommodate the middle ear mechanism to the varying amplitude of sound waves. The tensor tympani muscle is supplied by a branch of the motor division of the fifth nerve; the stapedius muscle by a branch of the seventh nerve. In many respects the neuromotor apparatus controlling the adjustment of these muscles is analogous to the accommodation mechanism of the eye, in which the ciliary muscle, innervated by the third nerve, automatically controls the focus of the crystalline lens, to maintain clear vision.

So far as we know, the action of these muscles is automatic. That a cerebral control can be exerted on this automatic mechanism is suggested by the following:

One of the writers is able at will to produce a roaring in both ears. Try as he will, he cannot produce this roaring in only one ear; in fact, he cannot make one ear roar louder than the other. This sound, produced by voluntary effort, consists of a rapid sequence of beats. There is a definite intermission between these beats. The beats and the intermissions are synchronous in both ears. There appear to be perhaps fifteen to the second; this is, of course, merely approximate, but there is a definite rhythm. The slightest effort produces this roaring at any time; it requires, however, a distinct effort to maintain the roaring. By concentrated effort the ears can be made to continue to roar for eighteen seconds; the roaring then ceases, but can be made to recur at once after cessation. A possible explanation of the phenomenon is the exercise of a voluntary control over the action of either the tensor tympani or the stapedius muscle or both.

ENDOLYMPH MOVEMENT.

The first problem of the vestibular apparatus is that of the physics of its end organ. Upon theoretical consideration, it has been generally assumed and regarded as more or less determined that the endolymph moves freely throughout all portions of the membranous labyrinth; that the endolymph movement in the semicircular canals acts upon the nerve receptors in the crista ampullaris of each canal; and that the direction of the endolymph movement determines the nature of the afferent impulse. The peculiar physical structure, and the configuration of the semicircular canals and utricle and saccule lends credence to this view. Given an organ made up of open tubes, placed in definite planes, and containing endolymph throughout their full extent, it has been thought obvious that this sense organ registers changes of position of the head by means of mass movement of the contained fluid. Doubt has been expressed as to the accuracy of this theory, especially concerning the possibility of endolymph movement within the membranous semicircular canals.

Question: Does the endolymph move freely within the membranous labyrinth?

Against: Physical considerations—

1. It would seem that the small caliber of the membranous semicircular canals must interfere with mass movement of the lymph within them, on account of capillarity.

2. It would seem possible that pressure variations, without mass movement of endolymph, can explain the origin of impulses from the end organs.

Physiologic considerations;

1. In fishes in which the semicircular canals and their ampullæ had been destroyed, S. S. Maxwell has produced all normal eye responses, except the horizontal, by mechanical stimulation of the otoliths of the utricle. This is an important contribution to the physiology of the internal ear. He further showed that the horizontal canal can be ligated or elevated into the vertical position, without interfering with the responses from turning in the horizontal plane. He points out that all the essential responses can be produced under conditions in which endolymph movement is impossible.

For: Physical considerations—

1. The physical arrangement of the semicircular canals is theoretically ideal for recording inertia of the contained fluid, from every possible movement of the head.

2. Maier and Lion (Arch. f. Ohren., Nasen u. Kehlkopfhlk., 107:149, Leipsic, May 20, 1921) have observed movement of the endolymph within the semicircular canals of living animals after caloric stimulation.

3. One of us has demonstrated mass movement of fluid within a capillary glass tube under conditions approximating those of the membranous semicircular canals. The lumen of the tube is between .3 and .4 mm.; the tube is semicircular and both ends terminate in a small reservoir or artificial utricle. The entire apparatus is filled with vinegar, which contains particles in suspension, easily observed under the microscope. With this tube in a vertical position and the reservoir uppermost, raising or reducing the temperature of one side is followed by circulation of the liquid; this can be easily observed under the microscope. The movement of the liquid begins slowly and gradually increases to a maximum velocity, which is maintained as long as the conditions of temperature in the tube are maintained. The movements of the liquid are always in the direction corresponding to the principles of the thermosyphon. During one observation of these caloric tests, one end of the tube accidentally became blocked; when heat was applied near the opposite end, two currents in opposite directions were observed at the same point within the lumen of the tube. In other words, free fluid movement was demonstrated in two different directions within a tube of the caliber of .4 mm. or less.

The above physical experiment would seem to show that capillary resistance does not prevent mass movement of fluid within a capillary tube, when the fluid is in free communication with a common reservoir through both ends of the tube.

Physiologic considerations:

1. It is perhaps possible to explain Maxwell's experiments without sacrificing our faith in the old tenets of endolymph movement. He has emphasized the importance of the otoliths, about which so little is known; and he has shown that certain eye movements can be produced in fishes, irre-

spective of endolymph movements within the semicircular canals. This, however, is not positive evidence that, under normal conditions, the endolymph does not move.

The eye movements in fishes, upon which these observations are based, do not compare in variety or delicacy with the responses elicited in the human being. When the horizontal canal of the fish was ligated, it is possible that the impact of the fluid acted on the sensory nerve endings in the ampulla, even though this movement could not continue throughout the entire course of the canal. A gross horizontal movement of the eyes could be obtained under these conditions; this, however, is qualitative and not quantitative. If, after ligating the canal, there could be produced an after-turning nystagmus of normal duration and amplitude, and after the caloric test a full response of normal intensity and duration, we would then have more positive evidence against endolymph movement.

Again, when the horizontal canal was elevated to the vertical position, it is not probable that the ampulla was drawn out of place, because it is directly adjacent to the utricle.

2. The argument that tension and not endolymph movement determines the response is not tenable; a change of tension without movement is impossible. If, after turning, there is a tension at one end of the utricle and a relaxation at the other end of the utricle, there must have been a displacement of the fluid content—in other words, mass movement.

3. In cases of fistula into a semicircular canal, pressure applied produces immediate responses. These responses cease promptly on the cessation of the pressure. There is evidently no prolongation of the response after such stimulation has ceased. After turning, the nystagmus and vertigo normally continue from 20 to 30 seconds. From this it may be concluded that the stimulus has persisted from 20 to 30 seconds after the turning has ceased. Immediately after turning, the nystagmus and vertigo are at their maximum. They gradually subside; the amplitude of the nystagmus becomes smaller and smaller, and the individual feels that he is rotating slower and slower, until he comes to a full stop. This is a common observation and can be demonstrated on any normal individual; it would seem that it can only be interpreted on the basis of a movement of the endolymph, which is at its maximum imme-

diately after the turning, and which gradually diminishes until it comes to a full stop.

4. A recent pathologic case bears directly on this subject (Georgie Johnson). In the examination of this patient, pneumatic pressure in the left ear produced:

(1.) Nystagmus, large amplitude, horizontal; slow component to the right; quick component to the left.

(2.) Past pointing of all extremities to the right.

(3.) No falling—all responses were in the horizontal plane. The nystagmus and past pointing appeared promptly when pressure was applied; continued while pressure was maintained; and ceased at once when pressure was discontinued.

These responses fulfill the conditions of the fistula test and indicate a fistulous opening into the left horizontal canal.

In contrast to the active responses from the fistula test, the left horizontal canal gave no response to turning or caloric stimulation.

Obviously the crista ampullaris of the left semicircular canal, with its sensory receptors, was functionating. The responses from its stimulation were prompt and qualitatively correct. They were elicited not only by air pressure, but also, as demonstrated by motion pictures, merely by touching the fistula region lightly with a cotton tipped probe.

It is of particular interest that, although the crista ampullaris was actively functionating, yet it was entirely unaffected by turning and douching. Obviously, something interfered with the function of the horizontal canal itself. The most reasonable explanation of these phenomena appears to be that there was an obstruction in the horizontal canal, posterior to the fistulous opening, which interfered with the endolymph movement within the canal, and which did not permit endolymph movement or impulse to affect the ampulla.

In Ewald's original experiment on pigeons, he plugged the horizontal canal just behind an opening in the canal. Pressure caused the eyes to rotate toward the opposite side. This case of Georgie Johnson closely resembles this experiment of Ewald. A similar case was shown by one of us before the American Otological Society in 1917—case of John Brooks. It happened that the fistula in this case was in the right ear and showed:

(1.) Nystagmus large amplitude, horizontal; slow component to the left; quick component to the right.

(2.) Past pointing of all extremities to the left.

(3.) No falling—all responses were in the horizontal plane. These two cases, Georgie Johnson and John Brooks, show all the phenomena of horizontal canal stimulation, by means of the fistula test. Johnson showed the normal responses to be expected on stimulation from behind the left ampulla, whereas Brooks showed the diametrically opposite responses to be expected on stimulation from behind the right ampulla. No positive deductions should be made from either of these cases; the only positive proof would be complete histologic examination, demonstrating an obstruction in the horizontal canal posterior to the fistulous opening. Short of histologic proof, however, these cases seem to be exactly analogous to the original Ewald experiment.

Resumé.—Considering the available evidence, the following conclusions are suggested:

1. Capillarity does not prevent endolymph movement within the semicircular canals.

2. Endolymph movement within the semicircular canals appears to be a factor in determining the character of the impulses from the end organ.

3. The otoliths play an important part in the detection of movement; apparently the otoliths and cristæ of the semicircular canals cooperate in function.

MECHANISM OF EXTRAOCULAR MOVEMENT.

The preceding problem concerned the physics of the end organ and the mechanism of the production of stimuli; the next problem is that of eye movement resulting from ear stimuli. Before attempting, however, to consider the vestibulo-ocular mechanism, it seems wise to discuss the ocular mechanism with which the ear is connected. We will therefore attempt to analyze the essential mechanism of ocular movement itself; then to follow the functional relationship that exists between the internal ear mechanism and this ocular mechanism.

In more primitive stages of the evolution of the ocular mechanism, one eye is placed on each side of the head and covers its own individual field of vision. In this stage each

eye may be moved independently. In higher forms, the two eyes are placed anteriorly, and to a large extent cover the same visual field. The fields are thus necessarily superimposed, and fusion of images is attained. As binocular single vision, normal to higher animal types, necessitates maintaining fusion in all possible directions of gaze, an adjustment of the visual axes must be maintained. Thus is evolved the conjugation of ocular movements—an automatic process. The term "associated ocular movements" signifies the unity of purpose of the two eyes, which are operated in such a manner as constantly to direct both visual axes toward the fixation point, in all possible directions of gaze. The association of ocular movements is not only automatic and involuntary, but is so firmly established as to defy voluntary effort to avoid it. Normal individuals cannot voluntarily rotate one eye in any direction in the slightest degree without also rotating the other.

By the very nature of the anatomic relations of the eyeballs and the extraocular muscles, it is obvious that each of the twelve extraocular muscles must play a definite part in every normal ocular movement. In each movement, each muscle acts as prime mover or synergist or control. Those twelve muscles can instantly and with micrometric accuracy direct the two visual axes toward any point within the range of the possible eye movements. This is true not only as regards direction of gaze but also as regards distance. The accurate degree of convergence of the axes, to correspond to the distance of the fixation point, is a part of the adjustment.

Manifestly, the nervous mechanism concerned with these movements includes at least three segments, viz.: (1) The peripheral or neuromuscular mechanism; (2) the cerebral projection tracts, and (3) an intermediate or coordinating mechanism.

1. The peripheral or neuromuscular mechanism is the final common pathway for all impulses causing ocular movement. This entire mechanism is called into action for each eye movement, whether voluntary or reflex.

Disturbance of any portion of this mechanism results in a proportionate loss of movement in terms of individual muscles or nerves—e. g., paralysis of the abducens or paralysis of the third nerve.

2. The cerebral projection pathway by which voluntary ocular movement is accomplished is a derivation of the pyramid tract.

It should be mentioned that fibers of the pyramid tract have not been histologically traced directly to the cells of the eye muscle nuclei. It should be also noted in this connection that pyramid fibers have not been traced to the motor cells of the spinal cord.

From the viewpoint of the cerebral motor mechanism, the eye movements conform to the general rule applying to all bilaterally grouped voluntary movements. They are not paralyzed, and are but slightly affected, by cerebral lesions which cause hemiplegia; either cerebral hemisphere is apparently capable alone of calling into action the complete range of normal extraocular movements. The usual interpretation assumes that there is a bilateral cortical representation for all muscles comprising such muscle groups.

We have frequently observed conjugate deviation of the eyes, from acute unilateral cerebral lesion or irritation, usually in association with convulsions or disturbance of consciousness. This conjugate deviation, however, never persists as a permanent symptom. Cerebral lesions do not interrupt the conjugation of eye movements. This is true, even following decerebration, if the midbrain is not injured.

3. The Coordinating Mechanism of Ocular Movement.—It is this coordinating mechanism, interposed between the cerebral and the peripheral segments, that commands our particular attention, since, according to our conception, it is upon this mechanism that impulses from the labyrinth play, to produce reflex eye responses.

In experiments on dogs and cats, Wilson and Pike (Phil. Tr. Roy. Soc., London, 803:127—169, 1912) noted conjugate ocular movements produced by ear stimulation (electric and caloric), after decerebration. Similar reflex conjugate movements were recorded after removal of the cerebellum.

These observations have been confirmed by others. In one of our experiments on cats, conjugate eye movements were observed after decerebration. A transverse incision had been made just anterior to the tentorium; necropsy later showed that this incision had been carried to the base of the brain and that the

cerebral hemispheres had been separated from the brain stem, with exception of a part of each occipital lobe and the posterior part of the optic thalami. Caloric stimulation of the right ear produced a few horizontal nystagmic eye movements; the amplitude was small and only a few movements were elicited, but the eye movements were definitely conjugated.

The significance of these observations is in the persistence of the congujation of eye movements after interruption of the cerebral or cerebellar connections. The type of movements observed requires the concerted, coordinated action of all the extraocular muscles. Since the cerebrum and cerebellum can thus be excluded, there remains only the brain stem in which to look for this coordinating mechanism. It would seem that this mechanism must include a system of neurons with fibers distributed to all the cells of the peripheral extraocular motor nerves; also that this mechanism is capable of sending to all these cells the correct impulses necessary to produce any accurately coordinated conjugate eye movement. This is also indicated by the variety of reflex eye movements observed after vestibular stimulation. It would seem probable that this coordinating mechanism not only receives afferent stimuli from various peripheral sources and directs the appropriate reflex responses, but that it is only by way of this mechanism that impulses reach the eye muscles from the cerebrum or any other source.

Clinicopathologic observations furnish important evidence concerning this mechanism. Many cases on record have shown inability to rotate the eyes in certain directions of gaze, although there was no paralysis of any of the extraocular muscles. These cases are designated as paralysis of associated ocular movement, to the right, or to the left, or upward, or downward, or in convergence. Such cases show lesions involving the posterior longitudinal bundle or the brain stem near the aqueduct of Sylvius. Cases of this type, showing a disturbance of associated eye movements without paralysis of any of the eye muscles, point definitely to lesions of the mechanism under discussion and indicate that the posterior longitudinal bundles and other association and commissural neurons within the brain stem are utilized by this mechanism.

According to the above conception, all eye movements,

whether voluntary or reflex, are under the control of this coordinating mechanism, which not only acts as a switchboard of distribution but also manifests a certain energy derived from its own neurons, similar to that of the Audion electric amplifier. This energy is distributed in a selective manner and results in conjugate movements.

The classic extraocular symptom of lesion of the peripheral neuromuscular mechanism is paralysis of individual ocular muscles; of the cerebrum, is conjugate deviation; of the coordinating mechanism in the brain stem, is disturbance of associated eye movements.

Many other coordinated neuromuscular mechanisms might be cited as analogies of the coördinating eye mechanism. For example, the respiratory movements are controlled by the "respiratory center" in the medulla oblongata. All movements of respiration are executed by voluntary muscles, each supplied by peripheral motor nerves; the whole series of these nerves arises from different levels, from the pons down to the lower dorsal segments of the cord. The respiratory center functions automatically through the influence of CO_2 in the blood. Voluntary control through cerebral impulse may step in and modify respiratory movement, by change of rhythm or temporary arrest. Reflexly, various afferent impulses may also step in, such as emotional reactions and application of sudden cold to the skin. It must be noted, however, that whatever impulse is transmitted to the respiratory center, the response is in terms of synchronous action of many muscles controlling the respiratory movements. Decerebration or removal of cerebellum does not interfere with the automaticity of the respiratory mechanism or with the synchronous and reciprocal action of the muscles it controls.

From the above consideration of the coordinating mechanism itself, we now turn to a discussion of its functions; how it is brought into action by stimuli from the cerebrum and from various sense organs.

Voluntary movements of the eyes, under normal conditions, consist of associated movements in all conceivable directions, except rotation in the frontal plane, and divergence. In close relation to the voluntary movements are certain instinctive automatic movements, in the production of which a cerebral

element is evident. Examples: Turning the eyes in the direction of objects suddenly coming into the peripheral field of vision; maintaining fixation of vision upon moving objects; rotation of the eyes in the frontal plane, which normally occurs when the head is tilted to one side; the necessary degree of convergence to attain fusion for objects at different distances from the eyes; rotation of eyes in the direction from which sounds are heard; or from which a touch is felt. This type of movement occurs instinctively, swiftly and accurately, without conscious effort, and in the interest of securing visual efficiency. Conjugation of ocular movements is always maintained.

These instinctive automatic movements tend to approach the purely reflex. In fact, it has been suggested by some that visual, auditory and tactile impressions may initiate ocular movements through the lower centers directly. There is some evidence that a direct reflex action can occur, but it is difficult to eliminate a cerebral element as a probable factor in most reflex ocular movements. There is, however, at our command one means of demonstrating a direct reflex movement of the eyes through lower pathways, by vestibular stimulation in decerebrate animals.

CONCEPT OF THE VESTIBULO-OCULAR MECHANISM.

Regardless of the exact physics of endolymph movement, the eye movements resulting from the turning and caloric tests are always in the exact plane of the endolymph impulse; further, the direction of the slow component of the nystagmus is always in the direction of the endolymph impulse. The almost mathematical precision of these reactions is familiar to all observers, irrespective of what theory is advanced to explain them.

Electric stimulation of the end organ, although a delicate test of function, is general and not selective. The caloric test is selective, but to a limited degree. The caloric effect can be applied only from the lateral aspect; therefore, the internal ear cannot be influenced in sagittal or oblique planes. It is the turning test that not only involves both ears but stimulates them in a manner identical to their daily physiologic activity.

The turning tests can be made to influence the internal ears in any plane desired. In this way, an infinite gradation in the

nature of the impulse can be sent inward from the end organ, depending only on the position of the head during the turning. Each gradation calls forth its own specific movement of the eyes.

That a reflex associated movement of the eyes in any predetermined direction or plane can be elicited at will by appropriate ear stimulation implies an accuracy of adjustment and intimacy of neural connections of the utmost variety and nicety. We must postulate not only an accurate gradation of end organ impulses but a like series of adjustments of the ocular coordinating mechanism. In each adjustment all twelve ocular muscles are concerned in an alternating series of actions and reactions.

Such phenomena can be explained only by the concept that impulses from the internal ear go to the ocular coordinating mechanism, which determines the appropriate ocular movements. By this concept, fibers from the vestibular nerve and nuclei are not distributed directly to the peripheral motor neurons of the third, fourth and sixth cranial nuclei, but to neurons of the ocular coordinating mechanism. This might explain the failure of attempts to trace the vestibulo-ocular tracts to the eye muscle nuclei, by means of secondary nerve degenerations.

THE QUICK COMPONENT OF NYSTAGMUS.

Question: Is it a cerebral function?

The usual concept of vestibular nystagmus has been that the quick component is of cerebral origin. There seems to be no question as to the origin of the slow component in the brain stem, but there has been nothing conclusive concerning the origin of the quick component.

For: The concept of the cerebral origin of the quick component is suggested by evidence such as

1. Voluntary fixation of the cerebral act. Deviation from the point of voluntary fixation occurs when the ears are stimulated. The explanation has been suggested that the quick component is an expression of the cerebral influence in momentarily overcoming the reflex pull from the ear.

2. During a vestibular nystagmus, gaze in the direction of the quick component amplifies the nystagmus, whereas gaze

in the direction of the slow component reduces it even to the point of complete disappearance. It might seem it is optional with the cerebrum to determine a maximum or minimum of amplitude, by the simple expedient of voluntarily reinforcing either the quick or the slow component.

3. Loss of the quick component under general anesthesia, also under other unconscious states, such as coma.

4. Case of Miss C. (Equilibrium and Vertigo, I. H. Jones, p. 392). On caloric stimulation of right vertical canals, there occurred a rotary conjugate roll of both eyes toward the right, no quick component to the left; stimulation of right horizontal canal produced conjugate deviation to the right, which persisted for the duration of the stimulus without any quick component to the left. Stimulation of the left vertical canals produced a normal rotary nystagmus to the right; left horizontal canal produced nystagmus to the right. The contrast was definite; the quick recoil to right was present and the quick recoil to the left was absent. Necropsy a few days later showed a large subcortical abscess in the right parietal region.

Against: On the other hand, the following arguments make it strongly suggestive that the quick component can be initiated by a lower mechanism, independent of the cerebrum.

1. The first argument given above (that the quick component is an attempt at cerebral fixation) is minimized when we note that the quick component is no less active when visual fixation is eliminated—by closing the eyes. Further, the intimate reciprocal relationship between the slow and quick components would seem to indicate the operation of a single mechanism rather than the cooperation of widely separated structures. On the basis of a cerebral origin of the quick component, we would have to consider the following sequence: Ear stimulation would act for the fraction of a second, whereupon a cerebral impulse would inhibit that from the ear and then substitute one of its own, effecting a reverse movement. This cerebral impulse, quickly subsiding, is then replaced by that from the ear, and this alternating series of reverse impulses continues for the duration of the stimulus from the ear. This appears less reasonable than a concept of the operation of a lower coordinating mechanism.

2. Absence of the quick component in general anesthesia or other unconscious states by no means proves its cerebral origin. The different degrees of disturbed consciousness are accompanied by the loss of not only cerebral activities but also by the loss of the various lower reflexes at different stages of the anesthesia.

3. In many cases of proven posterior fossa lesion, including cerebellar tumors and lesions of the brain stem, we have noted an absence of the quick component.

4. We have observed cases of proven pathology of the brain stem in which ear stimulation produced dissociated nystagmus —nystagmus of one type in one eye and of a different type in the other eye. It would be difficult to explain these two different types of quick components on a cerebral basis. When one sees, after ear stimulation, a horizontal nystagmus in one eye and a vertical nystagmus in the other eye, it seems more reasonable to conclude that the quick component originates in a lower mechanism, which automatically reacts to correct the particular deviation of each eye.

5. The conception of a lower automatic center which can produce the quick as well as the slow component, independent of the cerebrum, seems to be supported by recognized analogous coordinated reflexes. If acid is applied to the side of a decerebrate frog, the hind leg of the same side is moved in a manner to wipe off the irritant. This movement is not simply a flexion or an extension of the leg, but an orderly sequence of movements, including flexion and extension of several joints in a manner to accomplish a definite purpose. If the back of a decerebrate dog is rubbed, the socalled "scratch reflex" may be elicited, consisting of a series of quick to and fro scratching movements of one hind leg. In directing a stream of cold water against the inner surface of the pinna of a decerebrate cat, we have observed a quick coordinated to and fro movement of the head, characteristic of normal cats in attempting to dislodge irritants from the ear. In an analysis of the movements concerned in such coordinated reflexes in decerebrate animals, it is particularly to be noted that there is in each reflex a series of reciprocating to and fro movements, performed by groups of muscles. Many muscles are concerned in each reflex, and different groups are active in different

phases of the movements, alternating with remarkable accuracy and rhythm. These reflexes are of the nature of purposeful movements, and each of them can be initiated by volition. It is probable that they have evolved from purely voluntary acts to the stage of automaticity of the lower coordinating centers. As in vestibular nystagmus, some of these reflexes exhibit a series of to and fro movements, consisting of a principal movement and then a movement of recovery, in preparation for a repetition of the principal movement.

6. Some direct evidence has been presented from experiments on animals, indicating that lower centers may initiate the quick component of vestibular nystagmus. Barenne, cited by Wilbrant and Sanger (Neurologie des Auges, achter Band, die Bewegungsstörungen der Augenmuskeln, p. 308), observed no disturbance of nystagmus from turning in either direction, nor from caloric stimulation of either ear, in rabbits after removal of one cerebral hemisphere. Wilson and Pike, in 1911, observed spontaneous rhythmic nystagmus in a cat after destruction of the left labyrinth and the removal of the cerebral cortex of both sides. The optic thalami were intact in this experiment.

One of our experiments, although by no means conclusive, was not without significance. A cat, under ether, was decerebrated to the extent that most of the cerebrum was separated from connection with the brain stem. This was accomplished by an incision parallel and 1 cm. anterior to the tentorium, passing through the optic thalami. In spite of surgical shock, from which the animal did not recover, a few movements of both slow and quick components of nystagmus were elicited by caloric stimulation of the internal ear.

Resumé.—The quick component of vestibular nystagmus may be initiated by the lower automatic mechanism, independently of the cerebrum. However, cerebral impulses can modify the action of the lower mechanism.

SPONTANEOUS VERTICAL NYSTAGMUS.

Question: Is it indicative of lesion within the brain stem or involving the brain stem by adjacent pressure?

Against: 1. A small nystagmus in extreme position of lateral gaze is recognized as physiologic. Why should we not also

regard as physiologic a small vertical nystagmus, in the extreme positions, upward or downward, of vertical gaze?

2. We have observed in normal individuals a small vertical nystagmus upwards, after prolonged fixation in the extreme upward direction.

3. In patients showing general neuromuscular exhaustion, we have frequently seen prompt vertical nystagmus of fair amplitude, on extreme deviation of gaze, upward or downward.

4. Active vertical nystagmus, with the quick component upward, was observed by us in a cat upon recovery from ether, after injury of both internal ears. The right internal ear was first operated on; animal showed, after partial recovery from ether, a characteristic mixed rotary and horizontal nystagmus to the left, of eyes and head. The other internal ear was then destroyed. Immediately on recovery from ether, cat showed vertical nystagmus of each eye, synchronous, with quick component upward. Necropsy showed that there was no intracranial injury. (After many failures a technic was developed whereby the internal ear could be destroyed, through the external auditory canal, without hemorrhage and without entering the cranium.)

For: 1. Clinically, inflammatory processes within the internal ear produce a spontaneous horizontal nystagmus or a rotary nystagmus, or, most frequently, a mixed horizontal and rotary nystagmus. We know of no case of proven end organ lesion which has shown spontaneous vertical nystagmus (with the exception of the cat experiment noted above).

2. Brain stem lesions frequently exhibit spontaneous vertical nystagmus.

3. Cerebellar lesions, angle tumors and other lesions in the posterior fossa frequently show spontaneous vertical nystagmus. In our experience, vertical nystagmus has been observed in cases of posterior fossa lesion of the type of tumor or abscess —lesions producing local pressure. Others have made similar observations. However, that these lesions produce nystagmus not intrinsically but because of pressure, has not been regarded as conclusive. A recent case is illuminating on this point. The case was one of large cerebellar abscess. Among other phenomena, there was a large spontaneous vertical nystagmus upward. The abscess was drained. After operation the vertical

nystagmus was absent. Later, autopsy showed that the abscess was limited to the cerebellum. This case suggests that it was not the intrinsic cerebellar involvement that produced the nystagmus but the pressure upon the brain stem.

Resumé.—Spontaneous vertical nystagmus, per se, is not necessarily indicative of brain stem lesion, for the reason that it has been observed as a fatigue symptom. However, spontaneous vertical nystagmus has frequently been observed in patients with lesions in or near the brain stem and, so far as we know, has not been observed clinically in association with lesions in any other location. In patients who have other symptoms of intracranial lesion, a spontaneous vertical nystagmus indicates that the lesion is within or adjacent to the brain stem.

PERVERTED NYSTAGMUS.

Question: Is perverted nystagmus indicative of lesion within the brain stem, or involving the brain stem by adjacent pressure?

Definition: Perverted nystagmus may be defined as nystagmus of abnormal type resulting from vestibular stimulation.

We consider this phase of the subject of sufficient importance to describe the different types of abnormal eye responses to ear stimulation which we have observed, and to attempt a rough classification of perverted nystagmus:

1. Cases showing normal type of responses from stimulation of one ear but abnormal responses from stimulation of the other ear.

2. Cases showing abnormal responses from stimulation of both ears.

3. Simple perverted nystagmus—in which the eyes are coordinated, but the response is of a wrong type. Examples: Horizontal canal producing rotary, oblique or vertical nystagmus, instead of the normal horizontal; or vertical canals producing horizontal, oblique or vertical nystagmus, instead of the normal rotary.

4. Inverse nystagmus, in which the nystagmus is diametrically opposite to the normal response.

5. Convergent nystagmus, in which each eye shows nystagmus inward.

6. Dissociated nystagmus, in which the nystagmus of one eye is of a different type or direction from that of the other.

Slight deviations from the normal type of eye movements after vestibular stimulation may not be pathologic. Variations in the position of the head, or carelessness in other details of technic, may cause some variation in the responses. For these reasons a certain latitude of interpretation is necessary.

Against: 1. At each synapse in the course of nerve pathways there is probably a regrouping of the functional relations of the nerve fibers and a possibility of diversion of the pathways along which the impulse may pass. So long as an impulse is traversing one fiber, its course is fixed; at the synapse there may be several avenues, over each or all of which the impulses may continue. Every synapse in the course of the pathway increases the complexity of the pathways open to the impulse. Theoretically, if there is a block in any one segment of a pathway made up of several neurons, the impulse may deviate from its usual course by overcoming the threshold of collateral connections. On such a theory, Scarpa's ganglion might be the location where a lesion would divert the impulse from the horizontal canal to the pathways of the vertical canals. If this were true, it would be possible to account for a perverted nystagmus by a lesion not in the brain stem but within the internal auditory canal.

2. Even if the evidence were conclusive that perverted nystagmus is always of intracranial origin, it remains to be proved that it is indicative of brain stem involvement. Supratentorial lesions, merely by increasing general intracranial pressure, may cause various localized phenomena, such as papilledema or abducens paralysis. It is conceivable that such general pressure might so derange the vestibulo-ocular mechanism as to produce perverted responses.

3. In the absence of conclusive data as to the exact mechanism involved in the production of perverted nystagmus, and in the absence of a great number of well recorded cases, it is unwise to regard this phenomenon as a positive indication of brain stem involvement. To prove that it is not a positive indication, all that would be required would be one case of frontal lobe tumor (or any other lesion at a distance from the brain stem) which exhibited perverted nystagmus of any type.

For: 1. From our knowledge of the mechanics of the end organ and our interpretation of the normal physiologic responses, a perverted nystagmus is theoretically inconsistent with any disturbance of the end organ or eighth nerve.

2. The reflex pathway for normal nystagmus is from the vestibular end organ, through the eighth nerve and brain stem to the oculomotor nuclei. Theoretically, it would appear that perversion of the normal response must be due to a disturbance along this pathway; if the end organ and eighth nerve can be excluded, this disturbance would appear to be necessarily within the brain stem.

3. We know of no proven case of end organ lesion which has shown perverted nystagmus. Neither do we know of any case of the frequently observed neuritis of the eighth nerve which has exhibited this phenomenon. Tumors of the eighth nerve frequently exhibit perverted nystagmus; here, however, we have not a single eighth nerve lesion, but a tumor mass, often distorting the brain stem by pressure. It is a significant observation that in the tumors of the eighth nerve that we have seen, perverted nystagmus did not appear as an early symptom but came on later, presumably in the course of the enlargement of the growth.

4. Lesions of the brain stem itself frequently exhibit perverted nystagmus.

5. Lesions adjacent to the brain stem of the type causing pressure frequently show perverted nystagmus. It is sometimes noted that perverted nystagmus comes on in the course of the development of a posterior fossa lesion; as the lesion progresses the nystagmus becomes perverted.

6. We have no record of any proven case of lesion not adjacent to the brain stem which did exhibit perverted nystagmus.

Resumé.—Although this phenomenon has been observed for only a few years, and although the number of proven cases is limited, yet it may be said that a definite perverted nystagmus bids fair to be regarded as one of the most reliable signs of an involvement of the brain stem.

VESTIBULO-CEREBELLO-CEREBRAL TRACT.

Before considering whether the impulses producing physiologic vestibular vertigo pass through the cerebellum, we should

first consider the evidence from which the very existence of a pathway from the vestibular nuclei to the cerebrum has been assumed. The appreciation of movement is a concept that may be derived from various sensations, including visceral, tactile, muscle and vestibular sense. The normal recognition of movement is unquestionably a composite of sensations. In this composite of sensations the element that we are discussing is the vestibular sense. This we regard as a specific special sense.

We observe that from each type of stimulation of the end organ affecting the internal ear in various planes and directions, there occurs, in consciousness, a specific sense of motion. This sensation is definite in regard to direction, plane, duration, intensity and rate of movement—always corresponding to the plane of the stimulation. The normal individual, blindfolded and subjected to cold irrigation of the auditory canal, experiences a definite sense of movement; those in whom the vestibular organ is destroyed do not experience this sensation. In recent experiments we have noted the following: Normal cats, blindfolded and dropped, quickly right themselves in the air and land on the feet, whereas cats in which the internal ears have been destroyed, when blindfolded and dropped, fail almost completely to orient themselves in this respect. These movements were analyzed by slow motion pictures. Such evidence as the above would seem to indicate that impulses from the vestibular organ have the character of specific sensations.

It is generally accepted that there is a cerebral center for each of the other special senses; it would seem reasonable to postulate a similar cerebral representation for the reception of impulses from this end organ, which are recognized in consciousness. As to the location of such a center, there is no definite evidence.

Whatever its location, this vestibular center is to be regarded simply as the cerebral terminus for the reception of vestibular impulses—hence a sensory center. It is not an "equilibratory center." Equilibration is a complex motor process.

Question: Do the impulses from the internal ear, producing a sense of movement, pass through the cerebellum en route to the cerebrum?

Against: 1. So far as known, none of the other sensory pathways pass through the cerebellum. The visual and olfac-

tory pathways enter the cerebrum above the brain stem. Those of tactile, heat, cold, pain, pressure, kinesthetic, auditory and gustatory sensations, enter the cerebrum directly by way of the brain stem. Lesions of the cerebellum are never characterized by any loss or any impairment of these sensations. If, as expressed above, we consider that the impulses producing a sense of movement reach the cerebrum, from analogy of the other sensory pathways, it would seem improbable that the vestibular impulses traverse the cerebellum.

2. Although fibers have been traced from the vestibular nerve into the cerebellum, these may be accounted for as pathways concerned with functions not reaching consciousness. We have an analogy in the direct cerebellar tracts of the spinal cord, which enter the cerebellum via the inferior peduncles. According to the generally accepted view, these tracts, although they carry afferent impulses, are not concerned with sensations in consciousness. It would seem probable that the vestibulo-cerebellar fibers, like the direct cerebellar tracts, do not convey impulses concerned with the sense of movement, but constitute the afferent pathways for impulses to the cerebellum, resulting in coordinations of an automatic nature.

In consideration of these analogies, it might be inferred that the pathways conveying the impulses for the production of conscious sensations of motion from vestibular stimulation probably pass through the brain stem without any cerebellar connections.

For: 1. As before stated, fibers have been demonstrated to continue from the vestibular nerve into the cerebellum. Fibers also extend from the cerebellum into the cerebrum. This arrangement would seem to furnish an anatomic basis for the theory that vestibular impulses pass through the cerebellum en route to the cerebrum.

2. Each case of proven cerebellar lesion that we have seen has shown impaired or absent sensory responses from ear stimulation. The experience of many others has corrobated this observation.

Resumé: 1. It would seem unquestioned that motion sense impulses from the ear do reach the cerebral cortex. The location of the cortical center is not known. (2) Although it is by no means proven, such evidence as is available suggests

that these impulses traverse the cerebellum en route to the cerebrum.

CEREBELLO-PONTILE ANGLE TUMOR PHENOMENON COMPLEX.

During the past eight years we have observed the following phenomena in cases of angle tumor:

1. On the side of the tumor, a loss or impairment of auditory and vestibular function.

2. On the opposite side, normal auditory function, normal responses from the horizontal canal, but absence of responses from the vertical canals.

In brief, neurootologic examination showed a loss of function on both sides, except the cochlea and horizontal canal of the ear opposite the tumor.

In some cases, during the early period of development of the tumor, we noted merely the auditory and vestibular loss of function on the same side. In the course of weeks or months, one or more of the following additional phenomena were elicited:

1. The impairment and, later, loss of responses from the vertical canals on the opposite side.

2. A spontaneous vertical nystagmus has frequently been observed. In certain cases, this vertical nystagmus was not observed on first examination but appeared after an interval. After such a case has once shown spontaneous vertical nystagmus, we have never observed it to disappear, although we have noted variations in the amplitude of this vertical nystagmus.

3. In some cases, which had shown the complete phenomena complex, the horizontal canal on the opposite side had produced entirely normal nystagmus, vertigo and past pointing. In other cases, during the early period of development of the tumor, the horizontal canal of the opopsite side has shown normal nystagmus, but after an interval has produced perverted nystagmus.

4. In cases of well advanced growth in the angle, we have observed abnormalities in past pointing. This has been observed in a spontaneous past pointing, and also in an absence or the impairment of past pointing after ear stimulation. In some cases we have noted what we have termed a "crossed past pointing"; both arms would past point inward, or, both

arms would past point outward, regardless of the type of stimulation. For example, the patient would past point inward with both arms, regardless of whether he had been turned to the right or to the left.

It is difficult to interpret this phenomenon. In fact, our own experience to date has led us to draw no conclusions on the basis of abnormal past pointing alone. Abnormalities in past pointing have been observed in cases of cerebral lesion, as well as lesions within the posterior fossa; furthermore, we observed normal past pointing in a patient who proved to have a cerebellar tumor. Although abnormalities in past pointing do not appear, per se, to have anything approaching an exact significance or to furnish direct evidence in intracranial localization, it would seem wise, however, to make careful tests of the past pointing in all intracranial cases; abnormalities in past pointing can at times be of distinct value when correlated with other phenomena.

This phenomenon complex of cerebello-pontile angle tumor has been observed and corroborated by many others in this country. However, we have no knowledge of its recognition in other countries. The explanation of this phenomenon complex would appear to be as follows:

1. The tumor destroys the function of the eighth nerve on the same side by direct involvement of the eighth nerve.

2. The isolated loss of responses from the vertical canals of the opposite side; the occurrence of spontaneous vertical nystagmus; the occurrence of perverted nystagmus on stimulation of the opposite horizontal canal; and abnormalities of past pointing—all these phenomena would appear to be caused by pressure of the tumor upon the pons.

It is possible that an auditory phenomenon complex of angle tumor may become available, as suggested in the two cases referred to under "Auditory Problems," page 304.

CAUTIONS.

Experience has demonstrated the need of caution in drawing conclusions in cases of suspected angle tumors. The number of cases of cerebello-pontile angle tumors, carefully studied by neuro-otologic tests, is insufficient to establish a complete working basis for interpretation.

We have recently studied a case which shows that other pathology than tumor can produce certain otologic phenomena of angle lesion. Jesse Smith showed involvement of the third, fourth, fifth, sixth, seventh and eighth nerves, all on the right side. Ear tests showed complete loss of function in the right ear, both auditory and vestibular. Five days after the first examination auditory function began to return; within two weeks, under our direct observation, instead of complete deafness in the right ear, there was a such a complete restoration of hearing that he was able to hear a faint whisper, with the noise apparatus in the left ear. The caloric test also demonstrated the return of vestibular function within the same period of two weeks. The facial paralysis also disappeared. The neurologic diagnosis was multiple sclerosis. In this case it is to be noted, however, that there was no impairment of responses from the vertical canals of the opposite side.

In the earlier stages of angle tumors, the phenomenon complex is incomplete. Most assuredly, a diagnosis of angle tumor is not to be made, based simply upon abnormal or absent auditory and vestibular function in one ear. Given, however, a picture of absent auditory and vestibular function in one ear, impairment or perversion of responses from the opposite ear, with normal auditory function in the opposite ear—we have data indicating involvement of the angle.

Even under these circumstances, however, one cannot state that there exists a tumor which is confined to the eighth nerve and is probably removable. We have seen this phenomenon complex in tumor of the pons and tumor of a cerebellar hemisphere, invading the angle.

It is occasionally possible to draw valuable inferences from the history of such a case. If, over a considerable period, all the symptoms have been of the type referable to eighth nerve involvement, and then there begins to appear the absence of vertical canals' responses on the opposite side and other phenomena of brain stem involvement, we are justified in stating that the evidence suggests tumor of the eighth nerve.

The first symptoms of angle tumor are frequently those referable to functions of the eighth nerve; often it is the otologist who is first consulted by patients with this lesion. While the diagnosis of angle tumor is not dependent upon neuro-

otologic information, this phenomenon complex has been observed so frequently in proven cases that its presence may now be regarded as a valuable diagnostic sign.

NEURAXIAL DIFFERENTIATION OF THE FIBERS FROM THE HORIZONTAL CANAL AND THE FIBERS FROM THE VERTICAL CANALS.

Before discussing this problem, it is necessary to state exactly what is meant by this neuraxial differentiation. Our conception of this differentiation is that there is a divergence of vestibular fibers within the brain stem in such a manner that the fibers in relation to the vertical semicircular canals are separated by appreciable distance from those in relation to the horizontal canal.

This separation is considered to be sufficient to permit microscopic lesions to exert a selective effect upon one set of fibers and not upon the others.

Here it should be noted that the various vestibular responses have generally been described in terms of the semicircular canals, without recognition of the probable cooperation of the otolith organs of the utricle and the saccule in the production of these responses. The work of S. S. Maxwell has made clear that the utricle is a definite factor in the production of reflex ocular movements; we should bear in mind the probability of the participation of the otoliths of the utricle and saccule, to a greater or less extent, in the registration of movement in any plane. Therefore, although we are unable to estimate the exact relationship of the different nerve receptors, and consequently, for convenience, speak in terms of the semicircular canals, yet we should not lose sight of the part that may be played by the otolith organs.

Question: After entering the brain stem, do the fibers from the horizontal canal pursue a different course from those conveying impulses from the vertical canals?

Against: 1. No histologic proof of these separate pathways has been furnished. There are many vestibular pathways which have been traced histologically:

(a) Fibers from the vestibular portion of the eighth nerve have been traced to the socalled vestibular nuclei in the brain stem—the Deiters', Bechterew and triangular nuclei. These

nuclei occupy an area of considerable size and extent throughout the lower portion of the pons and the upper portion of the medulla oblongata.

(b) Fibers from the vestibular portion of the eighth nerve have been traced into the cerebellum without interruption.

(c) Fibers from the vestibular nuclei, by association and commissural pathways, have been traced in various directions, to the posterior longitudinal bundles into the spinal cord, and to the corpora quadrigemina of both the same and opposite sides.

All that is known of these nuclei and fiber tracts within the brain stem and cerebellum is that they bear some relation to the vestibular portion of the eighth nerve. No nucleus or separate fiber tract has been identified as being in relation with the utricle or saccule or any one semicircular canal.

2. Functional differentiation of fibers does not necessarily imply a divergence of their anatomic course. Pathologic processes may involve certain fibers, while others in close proximity escape. This may occur, even within a nerve trunk, as in degeneration of the papillomacular bundle of the optic nerve. The destruction of this particular bundle of fibers within the optic nerve has been histologically demonstrated in patients who have shown blindness of the central portion of the visual field. A similar selective process may occur within the eighth nerve; it is not a rare observation that in a neuritis of the eighth nerve, as in syphilis, the cochlear portion may be affected and the vestibular portion not affected, and vice versa. It requires no stretch of the imagination to conceive that within the eighth nerve there may be an involvement of the patricular fibers in relation to one semicircular canal, without the involvement of the rest of the nerve. Similarly, at any point throughout their intracranial course, it would seem that a pathologic process might affect certain fibers without affecting those even immediately adjacent.

3. The only evidence in favor of separate pathways from the different canals has been the clinico-pathologic. In the experience of all who have made vestibular tests of intracranial cases it has been the vertical canals that have shown impairment far more often than the horizontal canals. The nonresponse of the vertical canals should not be regarded as con-

clusive evidence that the vertical canals' fibers are widely separated from the horizontal canal fibers. It would seem reasonable to consider that the vertical canals' fibers may be less resistant; the horizontal canal mechanism is the more active, the more frequently used, and unquestionably the more primitive. It would seem that the horizontal canal mechanism might be more resistant, because of its being more firmly established in function than that of the vertical canals.

For: 1. The conception of separate neuraxial pathways from the different canals was advanced in 1916 by Mills and Jones, on the following data:

In a series of cases with intracranial lesions, in which the labyrinth and eighth nerves were normal, the vertical canals failed to produce responses.

These cases included lesions of the pons, angle tumors pressing against the pons, cerebellar lesions causing pressure against the pons, abscess of the fourth ventricle and internal hydrocephalus with pressure within the fourth ventricle.

These cases suggested the following:

If the lesions involving the pons caused a block in the responses from the vertical canals without impairing the responses from the horizontal canal, and as in all these cases the medulla oblongata was uninvolved, it was considered probable that the fibers from the vertical canals ascended into the pons, probably close to the floor of the fourth ventricle, whereas the fibers from the horizontal canal were presumably confined to the medulla oblongata.

Confirming this theory were the findings in a case of thrombosis of the posterior inferior cerebellar artery. This lesion, interrupting certain responses from the horizontal canal, causes softening of a portion of the right side of the medulla oblongata and part of the right inferior cerebellar peduncle. In this case all responses from all canals were normal, except that the right horizontal canal failed to produce vertigo and past pointing.

2. Much confirmatory evidence of the same character as the above has accumulated. W. P. Eagleton had previously noted that in the development of angle tumors the caloric test from the opposite ear gave negative responses; later we observed that under these circumstances, when the head was tilted back-

wards 60 degrees, stimulating the horizontal canal, the responses came through.

It is now a common observation that there is a nonresponse of the vertical canals in lesions of the posterior fossa involving the brain stem by infiltration or pressure. Few cases, however, have been seen in which the horizontal canal showed no response, when the vertical canals showed normal responses.

3. We have recently seen two cases, which, however, have not been confirmed by necropsy, in each of which the left horizontal canal failed to produce responses, whereas the responses from all of the other semicircular canals were normal.

4. Although no histologic demonstration has been made of separate pathways from the different nerve receptors in the end organ, yet there is evidence that the vestibular fibers spread out to be distributed to the vestibular nuclei. The fibers from the vestibular portion of the eighth nerve are for the most part interrupted in these nuclei, within a short distance from their entry into the brain stem. They enter the brain stem as a compact bundle, but their nuclear connections require a physical separation of all the fibers, preliminary to their termination. Complete ultimate physical dissociation of all these fibers must be accomplished, since each fiber has its definite destination in the vestibular nuclei, different from all others. These nuclei cover a large area; they involve the lower portions of the pons and the upper portion of the medulla oblongata, and extend from the lateral aspect of the brain stem well over toward the median line. Without speculation as to the ultimate course or destination of the neurons from the various portions of these extensive vestibular nuclei, the physical separation of the cell groups in these nuclei would in itself constitute a neuraxial differentiation of sufficient degree to answer the requirements of the conception given in the question, provided that functional grouping in terms of the semicircular canals exists in the vestibular nuclei.

Resumé.—While much remains to be learned of the intramedullary pathways and connections of the vestibular fibers, the evidence indicates that there is an appreciable separation of the neuraxial pathways of the fibers in relation to the horizontal canal from those in relation to the vertical canals.

SUMMARY.

Theoretical:

1. The available evidence suggests that there is a cortical area for the reception of impulses from the vestibular end organ.

It Is Probable:

1. That a cerebral cortical center for hearing is located in the temporal lobe. Also that each ear is in relation to the temporal lobe of each hemisphere.

2. That the otoliths and the cristæ of the semicircular canals cooperate in function.

3. That vestibular fibers are not distributed to the peripheral oculomotor neurons, but to the neurons of an ocular coordinating mechanism.

4. That the quick component of vestibular nystagmus can be produced by a lower mechanism, without impulses from the cerebrum.

5. That the impulses from the internal ear, producing a sense of movement, pass through the cerebellum en route to the cerebrum.

Reasonably Proven,

To be relied on in clinical work:

1. Capillarity does not prevent endolymph movement within the semicircular canals.

2. There is a coordinating mechanism for ocular movement within the brain stem.

3. Spontaneous vertical nystagmus, in patients who have other symptoms of intracranial lesion, indicates that the lesion is located within or adjacent to the brain stem.

4. Perverted nystagmus after ear stimulation is indicative of involvement of the brain stem, by infiltration or adjacent pressure.

5. Tumors of the cerebello-pontile angle frequently show a typical neuro-otologic phenomenon complex.

6. There is an appreciable separation of the neuraxial pathways of the fibers in relation to the horizontal canal and of the fibers in relation to the vertical canals.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday, November 10, 1924.

THE PRESIDENT, DR. E. P. NORCROSS, IN THE CHAIR.

Presentation of Specimens and Instruments.

DR. GEORGE W. BOOT exhibited a special instrument for the REMOVAL OF A GLASS BEAD FROM THE PHARYNX.

He claimed for it the advantages of simplicity, small cost and the fact that it can always be easily removed by unscrewing.

National Board of Examiners in Otolaryngology.

Among those present were the members of the newly organized NATIONAL BOARD OF EXAMINERS IN OTOLARYNGOLOGY, and these gentlemen were requested to explain the reasons for the foundation, and the objects, of this Board.

DR. GEORGE E. SHAMBAUGH pointed out that progress in medical science had gone on to such an extent that no one hoped to become really expert in more than a limited field, and this had resulted in the development of medical specialties to an extent that could not have been surmised a few years ago. The tendency toward specialization had outstripped all plans for providing proper training for such work, universities having been absorbed in the problems of undergraduate medical training. Physicians desiring to take up some special field have been left largely to their own devices for getting preparation. Some succeed fairly well in their efforts. The majority of men in recent years restricting their practice to otolaryngology relied solely upon a smattering of knowledge regarding a few operations, all of which they acquired in a few weeks' attendance on special clinics. These men lack entirely the training to make proper technical examinations and have acquired no appreciation of the indications for operative work, both of which are much more important than operative

technic in the preparation of the specialist. Such men cannot avoid doing a lot of unnecessary operating, which has brought untold harm to the specialty. This situation has been widely appreciated, and the subject of devising some correction has been often discussed.

He thought it would be quite logical to seek legislative measures to protect the public against the impostors posing as specialists without any proper preparation, but to secure correction by legislation seemed impractical. Two years ago the chairman of the Section on Otolaryngology of the American Medical Association asked for a committee to report on the question of devising some national examining board to pass on the qualifications of men who desire to be recognized as otolaryngologists, and Dr. Shambaugh was made chairman of a committee to look into the question. He secured the cooperation of the several national organizations devoted to otolaryngology and with a committee consisting of a representative from the American Laryngological Association, the American Otological Society, the American Laryngological, Rhinological and Otological Society and the American Academy of Ophthalmology and Otolaryngology, worked out a plan whereby each of these organizations, together with the Section on Otolaryngology of the American Medical Association, should appoint two members to act as the American Board of Otolaryngology. This report was favorably acted on by each one of the special organizations and the committee as recommended was formed.

The committee had met during the afternoon to work out organization. All of the members felt that the movement means very much for the elevation of the practice of otolaryngology. Dr. Shambaugh pointed out some of the advantages to those holding certificates from the American Board. First, anyone desirous of becoming a member of any of the four national organizations represented on the committee must possess the certificate before his name can come up for consideration. Second, any physician possessing the certificate of the American Board of Otolaryngology and who passes the Credentials Committee of the American College of Surgeons will be admitted to this organization without further examination. Third, the certificate will be in a form suitable for display in

the doctor's office and will serve as an introduction to the physicians as well as to the laity of the community in which he desires to locate. The possession of such a certificate will at once distinguish the properly prepared physician from the pseudo-specialist. Fourth, hospital managements in selecting men to do the work in otolaryngology will be glad to avail themselves of the information provided through the American Board of Otolaryngology in determining what men should be permitted to practice otolaryngology in the hospitals. The spectacle which one can witness any morning at many general hospitals, where a line of general practitioners, pediatricians and general surgeons is posted, each with his string of patients for nose and throat operations, reflects very little credit upon the hospital management which is permitting such a thing, and less credit upon the men who are indulging in this orgy of nose and throat operating. It is for the purpose of elevating the standards of practice in otolaryngology, for instructing the medical public in what constitutes proper preparation for those entering upon special practice and for protecting the general public against unqualified men attempting to practice in this field that this Board has been established.

DR. H. P. MOSHER (Boston) stated that the fathering of the Board belonged to Dr. Shambaugh, but that he was to hold the chairmanship for a year and then turn it back to Dr. Shambaugh where it belonged. He had not any experience with practitioners who wished to be taught otolaryngology in four weeks, evidently because they did not think he was bright enough to teach it in four weeks, so they went to Dr. Shambaugh.

It was very disheartening to him and to all of the men who had anything to do with the administrative affairs of the nose and throat departments in connection with the army to find that there were many men who used the specialty as a chance to get a "soft spot." As to the protection of the public, he thought that very beautiful, but believed the protection of the specialty itself to be more important. Many men have spent a great deal of time and tried to be decent in their specialty, and yet a number of men come in half trained and pose as specialists. This is not fair to the specialist or to the profession, and he thought this was one of the practical things that

should come out of the examination. The men on the Board were taking the matter very seriously and hoped to receive the cooperation of those practicing this specialty.

Dr. H. W. Loeb (St. Louis) thought that some of those present would remember when laryngology was little more than a spraying profession, but it had rapidly progressed and taken a place in surgery with spectacular results. Unfortunately, the specialty slowly acquired the balance which should go with the development of any branch of the medical profession, and in consequence 5,000 men are listed in the Directory of the American Medical Association professing to be specialists in otolaryngology, most of them thinking they are qualified. The members of the Board are going into the matter in a serious way, feeling that the Associations have deliberately selected them to bear the burden of determining who is qualified and who is not. They hoped that every member of the Chicago Laryngological and Otological Society would possess these certificates. The Board hopes that every qualified man in the country will have a certificate of qualification from the Board, and that they will be able to extend its influence to the general public so that much good will result from their labors.

Dr. B. R. Shurly (Detroit) stated that this Board is a development of the old school of otolaryngology, gradually and steadily progressing until it has become necessary for the specialty to classify and mark out what is properly considered best, what fundamentals should be required and what should be known. He hoped that everyone would get the proper spirit of the Board and realize that it is endeavoring to raise the standards, that every man properly qualified is to be given a chance and place. This standardization consists of three classes: (1) men who have been ten years or more in special work; (2) those who have been between five and ten years in special work, and (3) the younger men under five years in practice who are going into this special field and it is thought should be required to know more and to be better qualified than the young men of ten years ago. The Board will endeavor to have justice as its foundation and proficiency and high standards for its guiding stars.

Dr. W. P. Wherry (Omaha) reiterated what had been said regarding the sincerity of purpose of the members of the Board

and said that although he had been actively engaged in many different society affairs he had never entered into an organization in which there seemed to be such an intense appreciation of responsibility as is prevalent among the members of this Board. The fact that the work is to be put on in a most sincere, thorough and careful way would make these certificates that much more valuable.

He felt that the work of certificating applicants was just the beginning of the Board's functions, and that he could realize the advancement and elevation of this special field of practice to a higher plane. He hoped the day was not far distant when otolaryngology would be elevated to the high plane of a major specialty, where it should be. Only through organized effort, such as this Board is putting forth, could this ideal be accomplished.

Dr. F. R. Spencer (Boulder, Colo.) called attention to the fact that the Board should secure information from different medical schools of the country, particularly Class A medical schools, concerning the facilities of their schools for conducting graduate teaching in otolaryngology. This knowledge should then be classified and disseminated by the Board so that graduates from medical schools who contemplate limiting their work to otolaryngology will know where they can secure the best courses. He stated that it was not the purpose of the Board to grant a degree but to grant a certificate of proficiency in otolaryngology.

Dr. T. H. Halsted (Syracuse) said the Board in Ophthalmology had done much in its few years of existence to elevate standards in ophthalmology, and the same thing should be brought about in otolaryngology by the Board which had just had its organization meeting. Acting as one of the pioneers to place specialism on a higher plane of scientific medicine, this new Board of Otolaryngology should have an influence as well on the other specialties so that all may benefit. To succeed, the Board must have the sympathy and active support of the leading men in this branch of the profession, and he bespoke the hearty cooperation all present of the Society.

Dr. Joseph C. Beck said he knew that the interest of the Chicago otolaryngologists would be well taken care of by the

Board. The gentlemen had had some experience with this line of work in connection with the Board of the Academy, and several members of the Chicago Society had taken those examinations. He hoped that the examinations by the National Board would be stiffer.

THE CHAIRMAN expressed the thanks of the Society to the gentlemen for their explanations.

DR. ROSS HALL SKILLERN, Philadelphia, presented a paper on

"Some Observations on Certain Forms of Chronic Sinusitis."

DR. ROBERT C. LYNCH, New Orleans, spoke on the subject of

"Frontal Sinusitis."

with moving picture and lantern slide demonstration.

ABSTRACT.

DR. LYNCH stated that the procedure was not new, except in a few details, but was a modification of the Arnold Knapp and Jansen operations. Before proceeding with the operation the mouth should be carefully examined for defective teeth and these should be cleaned up. Such an extensive procedure would be totally unwarranted in an acute condition, but in dealing with a chronic state of infection there is time to prepare the patient for the procedure. The nose is cleansed by appropriate lavage, a postnasal plug is inserted and the nose is packed with strips of gauze soaked in alcohol. The packing remains in the unoperated side throughout the procedure. Just before making the incision the packing in the side to be operated is replaced by a strip of gauze soaked in adrenalin. The skin of the face is prepared with ether and alcohol, following which a 5 per cent solution of picric acid in alcohol is applied. The eyebrow is carefully cleaned but not shaved, and the incision is the same as for the Killian operation, except that it extends down through the periosteum. If the external projection does not go beyond the supraorbital notch it is not necessary to extend the incision to involve the supraorbital nerve. If the external limit of the sinus goes beyond the supraorbital notch the incision is extended to expose the external angle of the frontal sinus, and is extended down the lateral wall of the nose to within a very short distance of the end of the nasal

bone. Bleeding is controlled by clamping and ligating with No. 00 catgut. As soon as possible pressure on the eyeball is relieved. The periosteum is separated from the bone on the lower half of the incision by a big mastoid gouge. Where the periosteum dips into the fissure it is necessary to cut it loose with the sharp edge of a chisel. The lacrimal sac is dislocated from the lacrimal groove, being careful to keep along the periosteum, and the pulley of the superior oblique is dislocated, being careful not to buttonhole it. The periosteum on the upper half of the incision is not touched during the operation. The author's experience has been that it was necessary for these patients to return from two weeks to six months after operation and have a small spicule of bone removed, which this technic has eliminated. The separation of the periosteum continues over the common wall between the frontal sinus and the orbit throughout its entire extent. Traction sutures are placed through the periosteum of the orbit, giving a very good view, with apparently little disturbance to the orbital contents. The lowermost portion of the frontal sinus is taken off and the frontal sinus entered, a probe being used to determine the actual inside dimensions. Next they remove the bone which is represented by the orbital ridge, lying just under the incision. They proceed no higher on the frontal plate of the frontal bone than is necessary to obtain room enough to extend the removal of bone to the external angle. They extend the removal of bone outward until the external angle is completely obliterated. They then follow along the common wall between the orbit and the frontal sinus, and obliterate that angle throughout its entire extent, leaving no floor to the frontal sinus in any of its aspects. Next they take off the nasal process of the superior maxilla with a portion of the nasal bone and a portion of the lacrimal bone, which opens up the first portion of the ethmoid cell group. They follow that back and remove every vestige of the ethmoid labyrinth, which opens up the row of cells of the ethmoid. These are followed backward along the base of the skull and opened, the lower half at least removed and the mucous membrane thoroughly cleaned out. They remove even a portion of the perpendicular plate of the palate bone, opening to view the sphenoid sinus, which can be entered and broken down. Dur-

ing the past eighteen months, in the greater portion of these cases, they have been able to rather satisfactorily curet the major portion of the sphenoid sinus. They remove as much of the degenerated mucous membrane of the sphenoid sinus as can be reached and believe that one can only hope for a complete relief of symptoms by the most minute removal of every vestige of this mucous membrane.

In one patient operated four and a half years ago the eye seems to have receded slightly in the socket, with no disturbance to the patient. They get practically no deformity at all and so far have had no disturbance in eye function. By means of an X-ray picture showing the curet in the frontal sinus, with a long pair of forceps such trabeculæ as exist can be broken out under direct view. So far they have had to reoperate only one case.

Before the incision is made little cross cuts are made through the eyebrow to permit of proper approximation of the skin afterward. They do not suture the periosteum of the lower half of the incision to that of the upper half, but the sutures are placed with due regard to the cross cuts so that the orbital contents will preserve as nearly as possible their relation to the structures before they were cut into. At first a large rubber tube was introduced, but the size of the tube was gradually reduced, and in the last four operations no tube was used. Those four cases had no complications and no upsets. The after treatment is to let the patients severely alone. The large bandage is removed within thirty-six hours, and then the eye is exposed, with a small dressing over the incision. The patient is instructed not to blow the nose on the operated side and they do as little as possible intranasally following operation.

DISCUSSION ON BOTH PAPERS.

Dr. Norval H. Pierce thought the operation did not vary much from the old Killian, except that the frontal plate is not taken away, which meant better surgery, and he was sure that one could obtain as good a view into the frontal sinus, the ethmoids and sphenoids in this way as by taking off the external plate in the ordinary case. Where there is a fistula through the internal plate of the skull at the upper angle, it is

almost essential to take away the external plate. He could see no reason why Dr. Lynch should not have excellent results from this mode of treatment, except in those cases where the upper external boundary of the sinus is very high. In a large frontal sinus one has to take away nearly the whole orbital plate. He thought anyone who had dug into the sinuses where the blood was pouring out realized that it was not easy to remove the ethmoids and open into the sphenoid.

Dr. Pierce recalled a case which he operated upon about eighteen years ago that had been previously operated upon by Killian. It was a purulent recurrence. He could not find any nasofrontal duct or any opening into the cavity which bulged forward with its contents, proving that a recurrence is not necessarily to be regarded as a reflection on the operator or his technic. The adhesions that form may interfere with the drainage of the cavity, and he believed if Dr. Lynch should have a recurrence or two within ten to eighteen years this would not prove there was anything wrong with the operation. Such recurrence might be expected from any operation in this elaborate system of sinuses because of the unavoidable adhesions and scar tissue, whose formation cannot be prevented.

Dr. H. P. Mosher (Boston) said that the more he had to do with frontal sinus operations the more he felt that the failures are due to insufficient removal of the floor. In this procedure Dr. Lynch was focusing on the removal of the floor of the sinus, which Dr. Mosher considered the most important point in dealing with the frontal sinus operation. There are recurrences which are due in most part to failure to remove the outer angle, and also failure to recognize the frontal ethmoid cell, which is the most difficult to deal with. Dr. Lynch disregarded the pulley of the superior oblique and took it down, thus getting a better view of the posterior ethmoid cells, and Dr. Mosher thought they should feel indebted to him for bringing that point out. He considered the use of the gouge very unusual and believed the postnasal plug should always be put in by sight, not by touch. As to the removal of the frontal wall, Dr. Mosher had never had any trouble with sequestra from that. He agreed that it was impossible to remove the whole of the mucous membrane of the frontal sinus

by working from below, and saw no reason why one should not make a sufficient opening in the frontal to see what they were doing. He thought it entirely impossible to remove every vestige of the mucous membrane of the frontal sinus, the ethmoid and the sphenoid.

Dr. Charles M. Robertson said that the outer wall of the sphenoid cavity is a variable one, that it is wanting in quite a percentage of cases, or at least presents dehiscences of variable extent. When such a case is encountered an intimate relation exists with the internal carotid artery. In order to curet the sphenoid sinus then, with safety, it is necessary to enter the sinus at an angle of about 20° off the median plane, and this can be done more easily through the maxillary antrum, passing through the posterior ethmoid cells and keeping away from the outer sphenoid wall, which protects the integrity of the carotid. In one picture which Dr. Lynch showed, Dr. Robertson noticed that the curet was held parallel to the nasal septum, which, in his opinion, was absolutely dangerous, except in a very skillful operator's hands. If adopted by those less skilled he felt sure disastrous results would occur. He had seen cases recently in which there were two frontal sinuses which were small, each far out on the orbital arch, and accompanied by three very large frontoethmoid cells. One of these cells covered the entire frontal region as high as the hair line and communicated with the nose by a very narrow and long duct, which it would have been impossible to open by such an operation as Dr. Lynch had outlined, but he thought the operation was all right for the cases cited by Dr. Lynch and conducted by him.

In regard to removing the mucous membrane from the sphenoid through this procedure, he felt that it would be an impossible feat unless it were accomplished with a bent curet, and in that event one would not know when it was removed, because one could not see the entire sphenoid cavity wall, as the sinus extends to the median line. At times the sphenoid is divided into an upper and a lower sinus, in which event the lumen of the two cells would extend to the orbital wall on the opposite side of the head. The sphenoid sinus also is frequently found necrotic in its upper plate, and Dr. Robertson has seen two or three pituitary cysts extending into the

sphenoid cavity, and believed these cysts are sequelæ of sphenoid abscesses.

Dr. Alfred Lewy asked Dr. Lynch if he considered the removal of the posterior ethmoid group and the opening of the sphenoid necessary in all cases in which the external frontal operation is done, and also what he thought of local anesthesia for this operation.

Dr. Joseph C. Beck said that he recently had a case in which he thought this procedure was applicable, a case in which he had performed three external operations in three years. The patient was a young woman whose sinus extended beyond the hair line, externally on to the external canthus of the eye and posteriorly in two directions towards the optic foramen, and, separated by a high domed orbit, into two compartments. Each time he thought that he had removed all of the mucous membrane from every one of these recesses, but in each instance there was a recurrence. He thought that in operating along the line of Dr. Lynch's suggestion and taking away more of the lateral wall of the posterior ethmoid in this region he would get better drainage and final obliteration.

Dr. Beck thought that the procedure discussed by Dr. Lynch referred to the ordinary run of cases, and that if nothing else would relieve the patient this procedure was warranted, especially in women who do not wish to have deformities. There is a decided advantage in leaving the anterior wall alone if one can reach most of the mucous membrane and throw this cavity into the nasal cavity as one.

He asked Dr. Lynch whether there was not some epiphora for some time following operation, and some evidences of long continued diplopia.

Dr. Beck was much pleased with Dr. Skillern's clear way of outlining the pathology and the indications of treatment in different sinus diseases.

One point that has always irritated him is the use of air inflation in places where it cannot be controlled, and he believes that all necessary information can be obtained by suction in these cases.

Dr. Edwin McGinnis asked Dr. Lynch whether there are indications other than pain for this radical procedure. From the description of the technic and the pictures, it seemed to

him that nothing had been done intranasally to try to get drainage in this region, and he believed one could almost carry out the Lynch procedure intranasally, without doing the external operation. In a rather extensive experience he has not seen many pansinusitis infections and only occasionally has had to do a radical frontal operation.

Dr. Harry L. Pollock said that had several cases in which there was a dehiscence or necrosis of the posterior wall in which the carotid was exposed, and that he had drawn attention to this danger at the time of curetting. He has always been very careful about curetting into the sphenoid, and considers it a very grave mistake, especially to go in at a wrong angle, and believed it is much better to go in from the side.

Dr. Pollock thought the society should be thankful for Dr. Skillern's masterful description of the sinuses and the latest sinus work. He took just one exception to the statement that a long continued case in time necessarily shows a chronic or pathologic change in the mucous membrane. He has had two or three cases, one in which there were symptoms for four years, and one irrigation of the antrum cured the patient entirely, for there was a locked off antrum. In several cases in which symptoms have existed for a long time, opening them and obtaining aeration gave relief and there was not much pathology in the sinus. He agreed that a sinus which has continued to drain for a long time has a diseased mucous membrane, but believed there are exceptions.

Dr. George J. Musgrave suggested that instead of the technic outlined by Dr. Lynch, after reflecting the orbital periosteum one might enter the nose through the lamina papyracea, and with any punch forceps of the DeVilbis type remove the lacrimal bone. He uses a Wagner punch. The ascending process is at this point, an overhanging ledge, and the desired portion can easily be bitten off. It is then a simple matter to proceed upward, biting away the orbital wall and the ascending process to the T suture and as high on the frontal as is required.

Dr. Henry R. Boettcher stated that he visited Dr. Lynch and saw some of his cases about three years ago. Since that time he has operated on three cases. The first two were suc-

cessful. The last one developed a fistula at the second week, but after two weeks of irrigation cleared up. In all three cases there was a good deal of swelling of the orbital tissue, but after five days this subsided. In all cases only the anterior ethmoid cells were removed.

Dr. Robert H. Good thought that this operation was an excellent study and that some of the advantages were self-evident, but that the disadvantages should be stressed. The first disadvantage is that of removing the periosteum from the bone where the pulley of the superior oblique muscle is attached. The second is the removal of the entire floor of the frontal sinus, which allows the eye to retract backward and upward. The third thing to which he objected is that one does not obtain the proper view of the posterior wall of the frontal sinus, and that if necrosis of the posterior wall is found this cannot be removed by this route without great danger of injuring the dura. The most important part of a frontal sinus operation is the determination as to whether or not there is an extradural abscess, and it is necessary to have the best possible view of the posterior wall. The fourth point to which he objected is the removal of the lacrimal bone and lamina papyracea of the ethmoid, for the ethmoids can be removed thoroughly without sacrificing still more orbital wall.

One of Dr. Lynch's patients who had had this operation on the right frontal sinus had consulted Dr. Good. The man also had a left sided frontal sinusitis, which Dr. Lynch expected to operate on some time later. The patient found it absolutely necessary to wear a frosted glass over the right eye, and said he could not work without it. Dr. Good performed an operation, keeping the patient in the hospital one day, and sending him back to New Orleans in one week. He had seen the patient several times since then, and while there was no discharge from the sinus that he operated upon intranasally, there was still a discharge from the side operated on by Dr. Lynch. It had been two years since the original operation was performed, and the patient still found it necessary to wear a frosted glass over the right eye. Dr. Good did not see how diplopia could be avoided when the entire floor is removed and thought it a big mistake to interfere with the pulley. In his opinion, the conditions requiring external operation are

few, and it is surprising how large a percentage of these patients recover by simpler intranasal procedures.

Dr. Ross H. Skillern (Philadelphia), in closing, said he could not agree with Dr. Beck in regard to the needle puncture and the use of air, and that he would continue to blow air into the sinus, because he liked to know where the end of his needle was. If the air did not go in he does not force it, and agreed that if one continued to attempt to force the air and was underneath the mucosa there would be trouble. If one starts to blow and finds there is some obstruction, the procedure should be stopped to see what is the cause of the obstruction.

He thought the cases mentioned by Dr. Pollock, in which one or two irrigations effected a cure in long standing cases, were exceptions, and said that he might have seen cases like that which he mistook for subacute conditions and which also recovered.

Dr. Skillern stated that he had not given up the radical operation, but thought one should have an absolute indication for an extensive radical procedure, for instance, threatened cerebral complications, threatened orbital complications, fistula formation, or continuation of the foul secretion, and very severe persistent headache. In the first two instances he thought one should operate externally, whether there had been an internal operation or not. In the other two, one should see that the nose had been thoroughly cleaned out and if, by removing some possible obstruction within the nose, they could rid the patient of the trouble. Unless the symptoms are very severe, crying for something to be done quickly, he believed the patient should be given the benefit of the doubt, giving all the drainage possible. If he does not improve there is still time to go on to the radical work, and he proposed to carry out this plan in all cases hereafter.

Dr. Robert C. Lynch (New Oleans), in closing, said that he had learned several points which might change him somewhat in the future. He does not believe that any operation is suitable for every case that presents itself, but so far he has had none which he could not handle in this way. The operation is as difficult as anything one comes in contact with, and approaches nearer to major surgery than anything else one

is called upon to do in this department. They look upon recurrence as very likely, due to faulty technic, and are trying to avoid it as much as possible. One of the phases which has made him do the operation more often is that the more of these cases he comes in contact with the more he finds the ethmoid cells projecting over the eyebrow between the orbit and the frontal sinus, and it is mechanically impossible to reach these cells through the nose. He stated that he would never put in a postnasal plug again except by sight, but did not see why it was necessary to see the duct. He has never had any difficulty in finding it when the lower wall has been removed and the nasal process of the superior maxilla is taken off, for when one enters the lacrimal bone the duct comes into view.

As to curetting the sphenoid, he had mentioned this for the first time, because only in the last four or five cases has he been able to look into the sphenoid with satisfaction. He agreed that it is absolutely impossible to curet out every sphenoid sinus, even with an opening as wide as one obtains by this procedure, and that what curetting is done should be done only after a thoroughly good view is obtained. The idea of the curet being parallel to the septum was perfectly correct, because the view follows along those cells which lie closest to the attachment of the middle turbinate to the roof of the ethmoid. Following those straight back, one gets to the face of the sphenoid in a more wide open area than can be done by any other means, unless by one of the Moure operations which lay the whole face open. This procedure offers a better view over a broader area than by any other approach, either intranasally or extranasally, and at least more satisfactorily, because it can be sponged out and cleaned off so that one can palpate before going in.

Replying to Dr. Lewy, Dr. Lynch said he had stressed the fact that everything else had been done for these cases. If it was a true pansinus infection, then the posterior ethmoid cells are involved in the same process as the anterior and the rest of the cells. He thought it would be rather hard to imagine a chronic anterior cell ethmoiditis and not a posterior. In all the cases that are candidates for the operation the posterior ethmoid cells are removed and the sphenoid is opened.

Answering Dr. Beck, there had been no epiphora and no disturbance in the function of the lacrimal apparatus, so far as he had been able to see. They operated the one-hundredth case about three weeks before. Diplopia has occurred six or seven times, but has been cleared up at the end of three weeks.

The last case they operated upon was a mucocele. There was a chronic suppurative process involving all the cells, with marked exophthalmos of a chronic type. In that case there was diplopia for about five days, and similar cases have had diplopia which has gradually cleared up within a few days.

If the antrum is involved, they usually do the antrum operation first and take in the lower group of ethmoid cells, opening them up as completely as possible through the antrum, and going back to the anterior face of the sphenoid, and into the sphenoid through the antrum, thus offering the patient the possibility of clearing up that much infection and obviating the necessity for further operative interference. If that does not suffice, they do the radical frontal sinus operation as well. He thought Dr. Skillern gave the indications for the frontal sinus operation better than he could give them and agreed that it is absolutely impossible to do anything of that kind through the nose.

Answering Dr. Pollock, Dr. Lynch stated that they had used the curet in the sphenoid only in the last five cases, and did it with much fear and trembling.

The case mentioned by Dr. Good was one of three calamities they have had. One was their only death from meningitis, due to the fact that the patient had an infection of the frontal cavity and in attempting to prevent the scar they allowed him to develop an osteomyelitis, which extended into the meninges and caused his death. Now cosmetic results are considered less.

Dr. Lynch thought the case Dr. Good mentioned had a third nerve paralysis, but Dr. Good stated that this was not the case but that diplopia was present.

Dr. Lynch said they had one case with a colon infection, and in that instance had to open up the incision and drain through the external wound for quite a long time. There was paralysis of the third nerve as well as marked disturbance in the pulley function. The patient was living but has not much

use of the eye. In eighty or eighty-five cases, as many as he could get answers from, there was no disturbance of vision or in the pulley of the superior oblique. If this is buttonholed its attachment to the periosteum is loosened, which may interfere with its function.

The posterior wall, so far as Dr. Lynch's line of vision goes, is thoroughly open to view except possibly in a situation such as Dr. Pierce mentioned, which requires a special procedure.

He did not see any reason, with lack of overflow of tears, why the lacrimal bone should not be removed.

Their three cases of infection were the one with the third nerve paralysis, the one death from meningitis, and the third that of the man who went on a drunk and was struck in the eye and had an infection from that source.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

The December meeting of the Society was held on Monday evening, December 5, 1924, in the ballroom of the Auditorium Hotel, and was devoted to the celebration of the twenty-fifth anniversary of the foundation of the organization.

The Society was organized by Dr. E. Fletcher Ingals, in 1899, as the Chicago Laryngological and Climatological Society.

Preceding the dinner a group photograph was taken of all those present, and immediately following the dinner the Society was called to order by the president, Dr. Edward P. Norcross, who, in a few fitting remarks, installed two of the charter members, Dr. Norval H. Pierce and Dr. John Edwin Rhodes, as president and secretary, respectively, for the evening. Dr. Pierce had served as the fifth president, and Dr. Rhodes as the second secretary of the Society.

The charter members present, besides Dr. Pierce and Dr. Rhodes, were Drs. A. M. Corwin, George E. Shambaugh, Otto T. Freer, T. M. Hardie, James T. Campbell and Otto Stein.

The additional members present were Drs. George J. Musgrave, Jerome Strauss, Samuel Salinger, Ira Frank, Charles B. Younger, George P. Mosher, J. E. Lebensohn, S. J. Pearlman, Carl H. Christoph, George J. Dennis, Robert Sonnenschein, D. B. Hayden, B. F. Andrews, Thomas Faith, Alfred Lewy, Howard C. Ballenger, C. F. Yerger, Walter H. Theobald, Louis A. Ostrum, Elmer L. Kenyon, C. W. Geiger, Otis H. Maclay, George W. Boot, Harry Kahn, Charles M. Robertson, Harry L. Pollock, Joseph C. Beck, Frank Allport, Edward P. Norcross, Charles H. Long, George F. Fiske, T. W. Lewis, Frank E. Brawley, Robert H. Good, Dwight C. Orcutt, George T. Jordan, Frank Novak, Jr., and Austin A. Hayden.

The following addressed the Society:

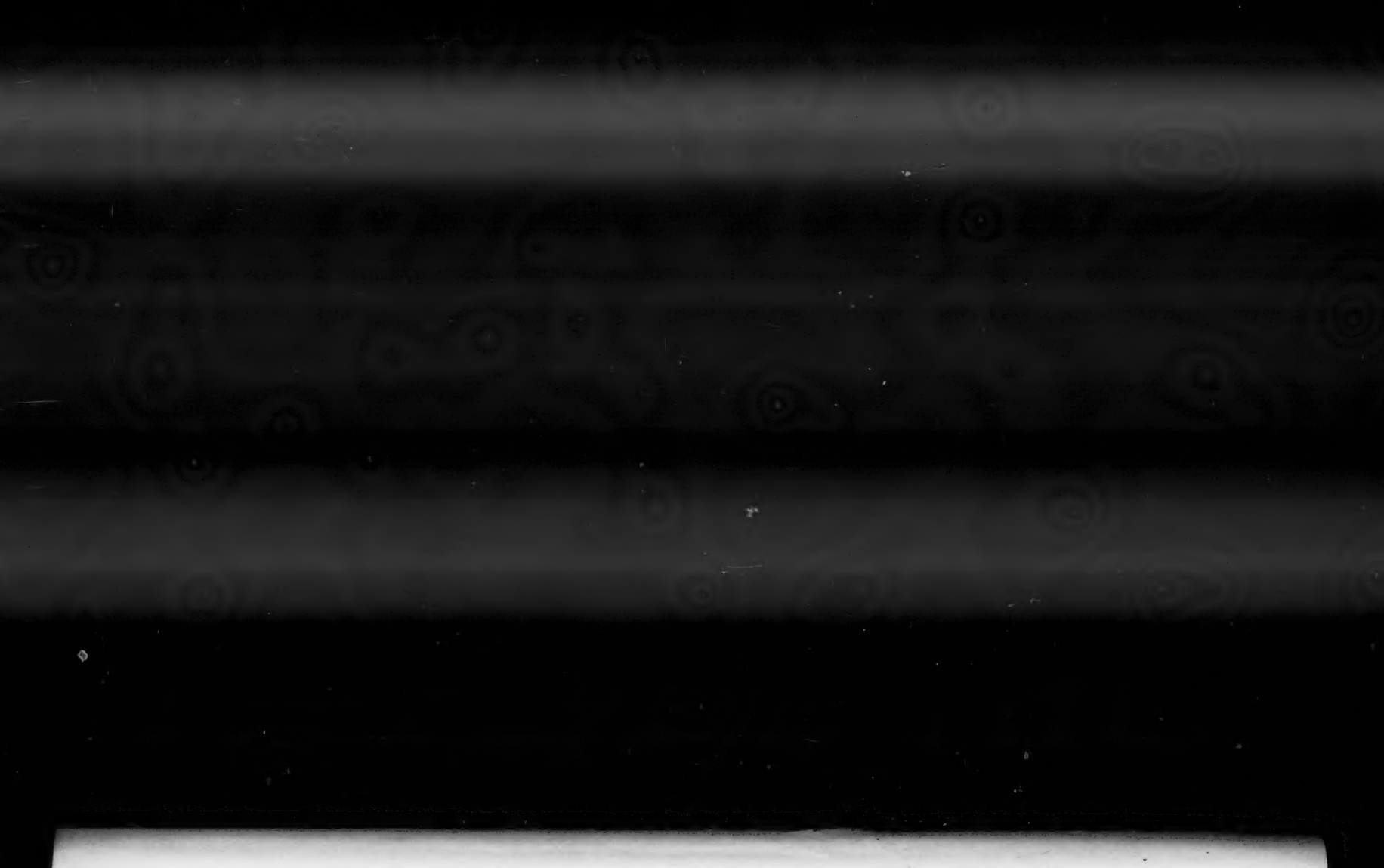
Dr. J. Edwin Rhodes, the first secretary, upon the organization of the Society. He stated that the first president was E. Fletcher Ingals, and the second, T. Melville Hardie.

Dr. Arthur M. Corwin, upon the personal characteristics of some of the members.

Dr. Frank Alport, upon reminiscences of the first ten years.

Dr. Wm. A. Evans, upon health standards.

Dr. O. T. Freer, upon the Society's value in constructive criticism of the views and practice of its members.

Drs. T. Melville Hardie, J. Holinger, Joseph C. Beck, E. L. Kenyon, upon reminiscences.

Dr. George E. Shambaugh, upon the future of the Society.